“STUDY ON THE ROLE OF ENDOSCOPY IN DYSPEPSIA SYMPTOM COMPLEX’’

Dissertation submitted

To

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI

With partial fulfillment of the regulations for the award of the degree of

M.S (General Surgery)

Branch-I

Government Kilpauk Medical College

Chennai- April -2015

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled 'STUDY ON THE ROLE OF ENDOSCOPY IN DYSPEPSIA SYMPTOM COMPLEX'' is a bonafide and genuine research work carried out by me under the guidance of Prof. P.N.SHANMUGASUNDARAM, MS, HOD of department of General Surgery, Kilpauk Medical College, Chennai-10.

This dissertation is submitted to **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI** in partial fulfillment of the degree of M.S. General Surgery examination to be held in **April 2015**.

Date:

Place: **DR M.VISAKAN**

CERTIFICATE

This is to certify that this dissertation is the bonafide work of

DR .M.VISAKAN

On

"STUDY ON THE ROLE OF ENDOSCOPY IN DYSPEPSIA SYMPTOM COMPLEX''

During his course in M.S. General Surgery from September 2013 to September 2014 at Government Kilpauk Medical College, Chennai-10.

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This is to certify that the dissertation titled **"STUDY ON THE ROLE OF ENDOSCOPY IN DYSPEPSIA SYMPTOM COMPLEX''**is a bonafide research work done by **Dr M.VISAKAN**, post graduate in M.S. General Surgery, Kilpauk Medical College, Chennai-10 under my direct guidance and supervision in my satisfaction, in partial fulfillment of the requirements for the degree of **M.S. General Surgery**.

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INTRODUCTION

Dyspepsia is not a diagnosis but it refers to a group of symptoms related to the pathology of upper gastrointestinal tract[1]. According to American gastroenterological association dyspepsia refers to chronic and recurrent pain or discomfort centred in the upper abdomen.Rome IIIcriteria for dyspepsia must include one or more of the following

- postprandial fullness
- early satiation
- epigastric pain
- epigastric burning

A clear diagnosis based on these symptoms will be difficult. Many diseases cause dyspepsia namely gastric and duodenal ulcers,oesophagitis,gastric and pancreatic cancer, gall stones etc. But in majority of patients with dyspepsia no clear pathologic cause can be determined and it is known as functional dyspepsia.

Upper gastro intestinal endoscopy is the most sensitive test for patients presenting with dyspepsia. But when taken into account, inthe large population of dyspeptic patients endoscopy cannot be done practically for all of them. It is even more difficult in developing countries like India where diagnostic facilities are limited.

There is no standard age cut off for patients above which endoscopy is indicated. So this topic was undertaken in Kilpauk medical college in Chennai to study the role of endoscopy in management of patients with dyspepsia symptom complex.

AIM OF STUDY

1.To study the various endoscopic findings in dyspeptic patients and to study the usefulness of endoscopy in patients presenting with dyspepsia with or without alarm symptoms

2. Incidence of H pylori in patients with dyspepsia

3. Incidence of oesophageal and gastric cancer in patients presenting with dyspepsia

REVIEW OF LITERATURE

Dyspepsia is derived from two Greek words: dys-poor and pepsin-digestion. There is no standard definition for dyspepsia. Dyspepsia symptom complex include a number of vague symptoms like upper abdominal pain, epigastric burning,early satiety or fullness,heart burn, nausea and regurgitation. The alarm symptoms when serious pathology should be considered include

-any persistent dyspepsia in patients above 55 years of age

-weight loss

-gastro intestinal bleeding

-persistent vomiting

-dysphagia

-epigastric mass

-previous gastric ulcer

- iron deficiency anemia

-gastric surgery

-suspicious barium meal

-anti inflammatory drugs usage

-hospitalisation for epigastric pain

Dyspeptic patients can be broadly divided into two subtypes-organic or pathological and functional. Most of the patients fall into the second subtype.ROME III criteria for functional dyspepsia include the above said symptoms-early satiety,epigastric pain or discomfort and postprandial fullness with no evidence of structural disease. In western countries nearly 15% of population is affected by functional dyspepsia[2]. Though only 1 in 4 persons with dyspeptic symptoms consult the doctor, it possesses a considerable clinical burden due to high incidence,more so in developing countries[3].

Dyspepsia patients can be divided after endoscopy into

-ulcer like

-dysmotility like

-reflux like

- nonspecific groups

CAUSES OF DYSPEPSIA

LUMINAL GI TRACT

- Peptic ulcer disease
- Gastroesophageal disease
- Gastric or oesophageal neoplasia
- Gastroparesis(eg. DM, post-vagotomy, scleroderma, chronic intestinal pseudo obstruction, post-viral,idiopathic)
- Infiltrative and inflammatory gastric disorders (eg.Crohn's disease,eosinophilic gastroenteritis,sarcoidosis,amylodosis)
- Gastric infections (cytomegalovirus,fungus,TB,syphilis)
- Parasites (Giardia lamblia, Strongyloides stercoralis)
- Chronic gastric volvulus
- Chronic gasrtric or intestinal ischemia
- Food intolerance
- Irritable bowel syndrome
- Functional dyspepsia

MEDICATIONS

- Ethanol
- Gemfibrozil
- Estrogens
- Glucocorticoids
- Colchicine
- Iron
- Aspirin (other NSAIDs including COX-2 selective agents
- Digitalis preparations
- Levodopa
- Narcotic
- Niacin
- Nitrates
- Orlistatin
- Potassium chloride
- Quinidine
- Sildenafil
- Theophylline

Pancreatico biliary disorders

• Biliary pain (cholelithiasis ,choledocholithiasis, sphincter of Oddi dysfunction)
• Chronic pancreatitis
• Pancreatic neoplasms

Systemic disorders

• Myocardial ischemia
• Congestive cardiac failure
• Diabetes mellitus
• Thyroid disease
• Hyperparathyroidism
• Intra abdominal malignancy
• Pregnancy
• Renal insufficiency

EPIDEMIOLOGY

The prevalence of dyspepsia according to the various world wide population study is around 25% (range 10-40%) for a 3-12 month period[4]. But if heart burn or regurgitation are excluded, prevalence rate will be only around 3-15 %[5]. In less than 50% of the patients symptoms resolve over time according to the longitudinal studies[6].

The prevalence of dyspepsia is higher in women than men. This difference decreases as the age increases. New onset dyspepsia will be seen in approximately 5% of population each year[7].

In Mumbai, study was conducted which reported that 33.3% of the population had dyspeptic symptoms and significant symptoms was present in nearly 12% of the population[8]

Prevalence[9] based on the symptoms include

-reflux symptoms 25%

-dyspepsia without reflux symptoms 15%

-irritable bowel smptoms 15%

-GERD 10%

According to the study conducted by Breslin NP et al 'Study on gastric cancer and other endoscopic diagnosis in patients with dyspepsia without alarm symptoms' among 2867 patients under 45 years only 3 cases of gastric cancer was dignosed[10].So they concluded that doctor should consider endoscopy only for persistent or recurrent symptoms and not for all dyspeptic patients. Study conducted by Vakil N in 2741 patients also yielded the same result[11].

Lieberman D et al conducted study in endoscopic results in 99558 patients among which dyspeptic patients are 43% and among dyspeptic patients 36.5% are less than50 yrs without alarm symptoms[12]. They found that gastric cancer was related to increasing age,male sex,North American and Asian race.

According to the study conducted by Choomsri P et al in 291 patients only 19% had significant endoscopic findings[13]. More importantly, alarm symptoms was not related to endoscopic findings.30 among the 130 patients who were biopsied were positive for H pylori[14]. Similarly, H pylori infection was not associated with significant endoscopic findings. They concluded that dyspeptic symptoms were not associated significantly with endoscopic findings and hence empirical anti-acid therapy should be given first before subjecting the patient to diagnostic endoscopy.

In 50 patients with dyspepsia, endoscopic findings were studied by Khan N et al. In this study there were 35 males and 15 females and 41 patients were in the age group of 30-50 years. Epigastric discomfort was present in 90% of the cases followed by heart burn in 72% of cases. In half of these cases(50%) there was no significant endoscopic findings. Esophagitis (12%) was the most common endoscopic finding followed by gastric ulcer and duodenal ulcer in 10% and 8% respectively[15]. Carcinoma stomach was diagnosed in 1 case (2%). They also concluded that dyspeptic symptoms was not significantly associated with endoscopic findings.

Goh K L et al studied the association between H pylori and gastrointestinal disease. They also compared rapid urease test with culture, gram staining and histology. They concluded that rapid urease test, with 99.3%positive predictive value and 96.2% negative predictive value, is a reliable and rapid test for diagnosis of H pylori infection[16].

A study was conducted by Saba Fakhrieh about evaluation of endoscopic findings in Iranian patients with dyspepsia. Study was conducted over a period of 14 months in 940 out patients with dyspepsia. On comparing ulcer dyspepsia with non ulcer dyspepsia, ulcer like dyspepsia was common presenting in 69.3% of the patients under study. Peptic ulcer was the most common finding in ulcer like dyspepsia patients presenting in 133 cases (14.1%). Risk factor such as alcohol and

smoking was associated with peptic ulcer patients significantly.H pylori was present in 68.4% of peptic ulcer patients and 41.5% of patients with non ulcer dyspepsia[17].

Thomson A B R et al did endoscopy for 1040 patients with dyspepsia.603 cases among 1040 cases showed significant endoscopic findings. Oesophagitis was present in 43% of the cases which was the most common finding and peptic ulcer in 5.3% cases was the least common finding. Alarm symptoms was present in only 2.8% of the cases. Most of the patients had multiple dyspeptic symptoms. Nearly 45% of cases had ulcer like symptoms followed by reflux like symptoms (38%) and dysmotility like dyspepsia(18%)[18]. But endoscopic findings did not correlate well with endoscopic findings. In patients with reflux like symptoms,oesophagitis was the most common finding.H pylori infection was present in 301 cases and endoscopic findings were positive in many of these cases. It was concluded that the dyspeptic symptoms have little value in predicting endoscopic findings. This study similar to previous studies conclude that patient should be treated with acid suppressive therapy and endoscopy should be considered only in persisting symptoms in case of uncomplicated dyspepsia.

A study was conducted by Nowshad Khan et al to find the common causes of dyspepsia in Peshawar, Pakistan. Study was conducted for 6 months in 50 patients. Most of them were male in age grup of 30 - 50 years. Epigastric pain with discomfort was the most common symptom presenting in 90% of the patients.no significant endoscopic findings were noted in 25% of the population[19]. Among the endoscopy positive cases oesophagitis was the most common finding seen in 12% of the cases followed by gastric ulcer in 10% of the patients. Carcinoma stomach was present in 2% of the cases.

Among 254 patients studied by Singh V et al patients, who attended the out patient department 147 patients were subjected to endoscopic biopsy for histopathology. Among the 147 cases, 80 were symptomatic and 67 did not have any symptoms. Among symptomatic patients 61.3% were positive for H pylori and 56.7% of asymptomatic were positive for H pylori infection[20].H pylori was positive in 11 out of 13 cases of peptic ulcer.

A study was conducted by Yasmeen Khan et al to evaluate endoscopy findings in patients with symptoms of dyspepsia. It was conducted in Chhattisgarh, India. It was a retrospective study conducted on 593 patients for a period of 6 months. All patients with dyspepsia who underwent endoscopy were included in the study.64.9% of the patients were male and females were 35.1%. Mean age was 42 years. In this study, gastritis was the most common endoscopic finding and gastric

ulcer patients(6.7%) were more common than duodenal ulcer patients(4%)[21]. Gastric carcinoma was present in 4.6% of patients and oesophageal carcinoma was present in 3.7% of patients. Normal endoscopic findings were seen in 40% of patients.

Study conducted by Dalaney et al about the cost effectiveness of endoscopy compared to first treat and then test policy in patients over 50 years of age suggest that initial endoscopy is cost effective than usual management[22]

Sumathi et al did a study on 'Appropriateness of indication for endoscopy in India'. Among 3432 south Indian patients with dyspepsia for whom endoscopies were done 18.3%(284 patients) were diagnosed with malignancy and they were between the age group of 25-45 years. They concluded that the cut off age for malignancy in male as 38 years and 43.5 years for males.

Uehara et al –a total of 32,388 dyspeptic patients were included and 285 gastric cancer cases were detected among which 45 cases were without alarm symptoms. They concluded that cut off age for gastric cancer in dyspeptic patients without alarm symptoms should be above 40 yrs of age[23].

According to study conducted by Casburn-Jones AC et al among 135 patients diagnosed to have gastric cancer in age group above 55 years only 8 cases were without alarm symptoms and among them only 1 patient was in the curable stage[24].

So according to this study, even in patients above 55 years endoscopy does not significantly reduce the mortality rate.

Gillen et al following his study on role of endoscopy in dyspeptic patients found that gastric and oesophageal malignancies are extremely rare in dyspeptic patients below 55 years and also if malignancy is diagnosed they are usually in incurable stage[25]. Hence there is no indication for endoscopy for all dyspeptic patients below 55 years without alarm symptoms.

Marmo et al found that age limit for endoscopy should be decreased for males and increased in females to reduce the rate of missing gastric cancers. They concluded that age along with gender should be considered for deciding on the age limit for endoscopy .

Williams et al concluded that young patients with dyspepsia without alarm symptoms should not be over investigated and should be treated with acid suppressive therapy.

Christie et al in their study found that only < 1% of cases were diagnosed with gastric cancer in age less than 55 years and hence age limit for endoscopy in dyspeptic patients without alarm symptoms can be 55 years[26].

According to the study conducted by Phull PS in Scottish patients gastric malignancy were uncommon in age less than 55 years and if they rarely occur, they

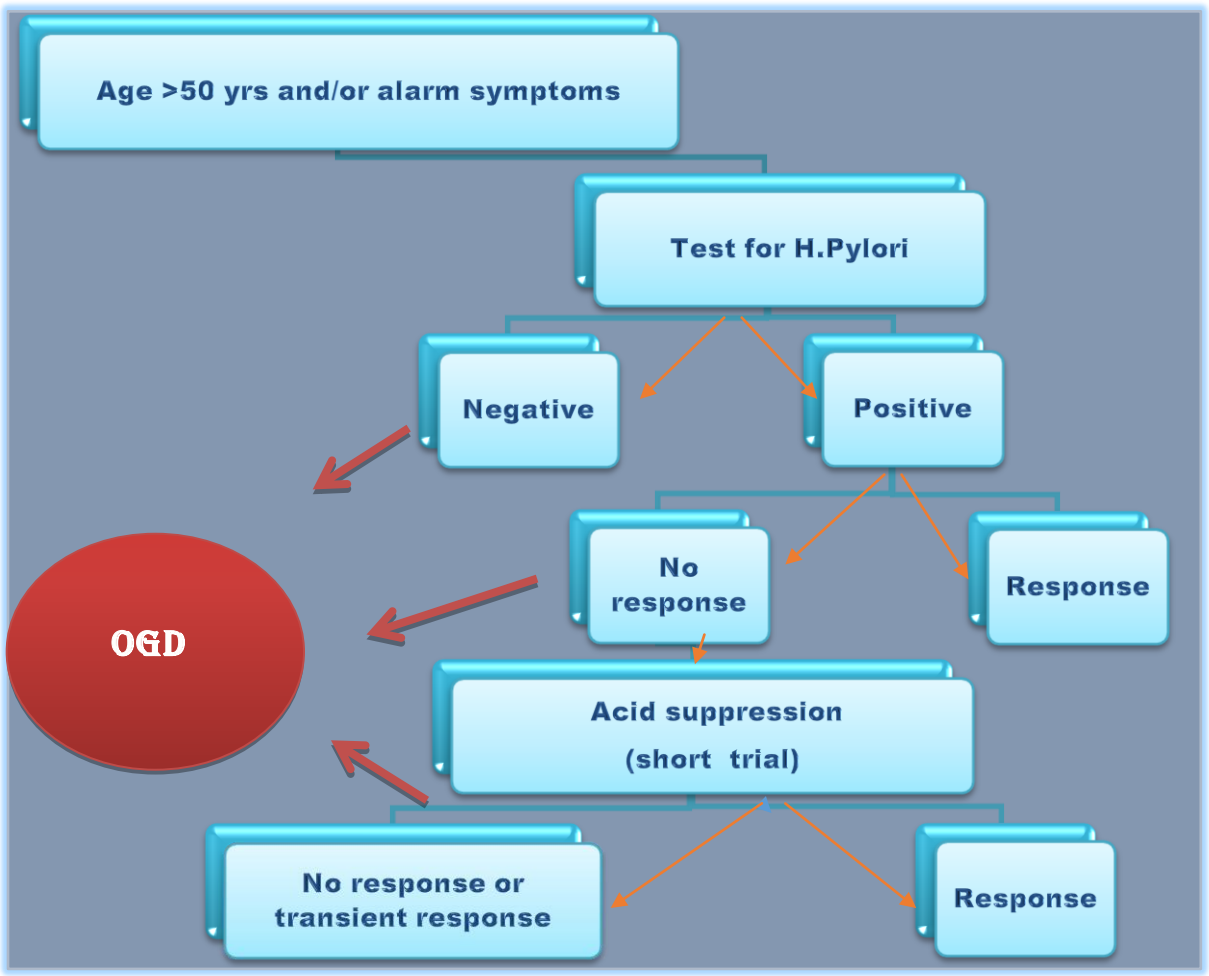
present with alarm symptoms. So raising thecut off age from 45 to 55 years for endoscopy for dyspeptic patients does not significantly affect the outcome[27].

Mimica M et al did a study to find out the cut off age for gastric cancer in developing countries (Herzegovnia-Bosnia). A prospective study was done for 4 years and found that if cut off age for endoscopy is 50 years for male and 60 years for female then the work load can be reduced to 50% without significantly affecting the outcome[28].

A Study was conducted byLiou JM et al in Taiwan where the incidence of gastric cancer was high. It was concluded that if cut off age was fixed at 45 years nearly 5% of the gastric cancers were missed. So the cut off age of 40 years for both genders would be optimal in places were incidence of gastric cancer was high.

According to the studyconducted by Wai CT in Asian patients in Singapore, among 5,066 patients 14.6% had peptic ulcer,5% had oesophagitis,0.47% had gastric cancer and 0.06% had oesophageal cancer.Gastric cancer was noted in 1.15 of 1000 in patients less than 45 years and 9.60 of 1000 in patients above 45 years of age.They concluded that 45years as cut off age for endoscopy in simple dyspepsia in Singapore and it varies depending on the local incidence of gastric cancer[29].

EVALUVATION OF DYSPEPSIA



EPIGASTRIC PAIN SYNDROME

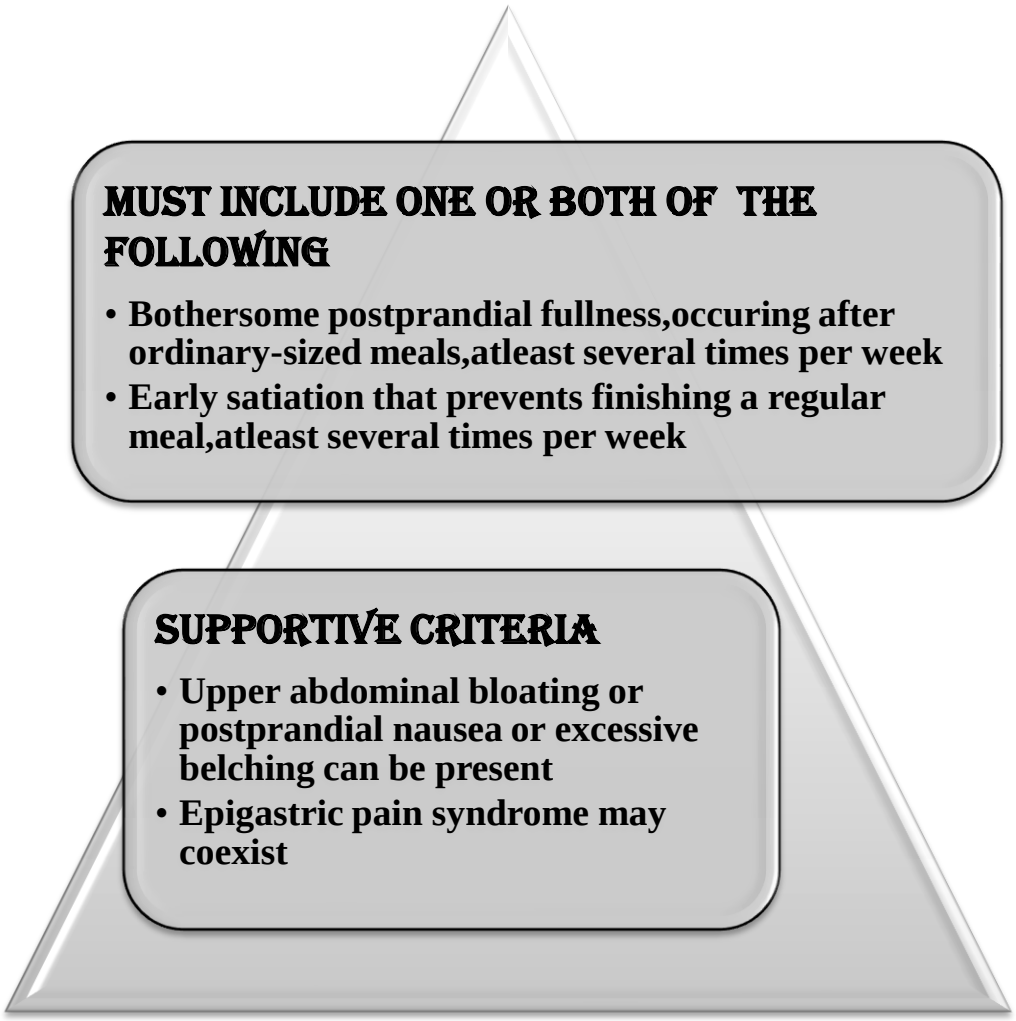
MUST INCLUDE ALL THE FOLLOWING

- **Pain or burning localized to the epigastrium of atleast moderate severity,at least once per week**
- **Pain is intermittent**
- **Not generalized or localized to other abdominal or chest regions**
- **Not relieved by defecation or passage of flatus**
- **Not fulfilling criteria for gallbladder or spincter of Oddi disorders**

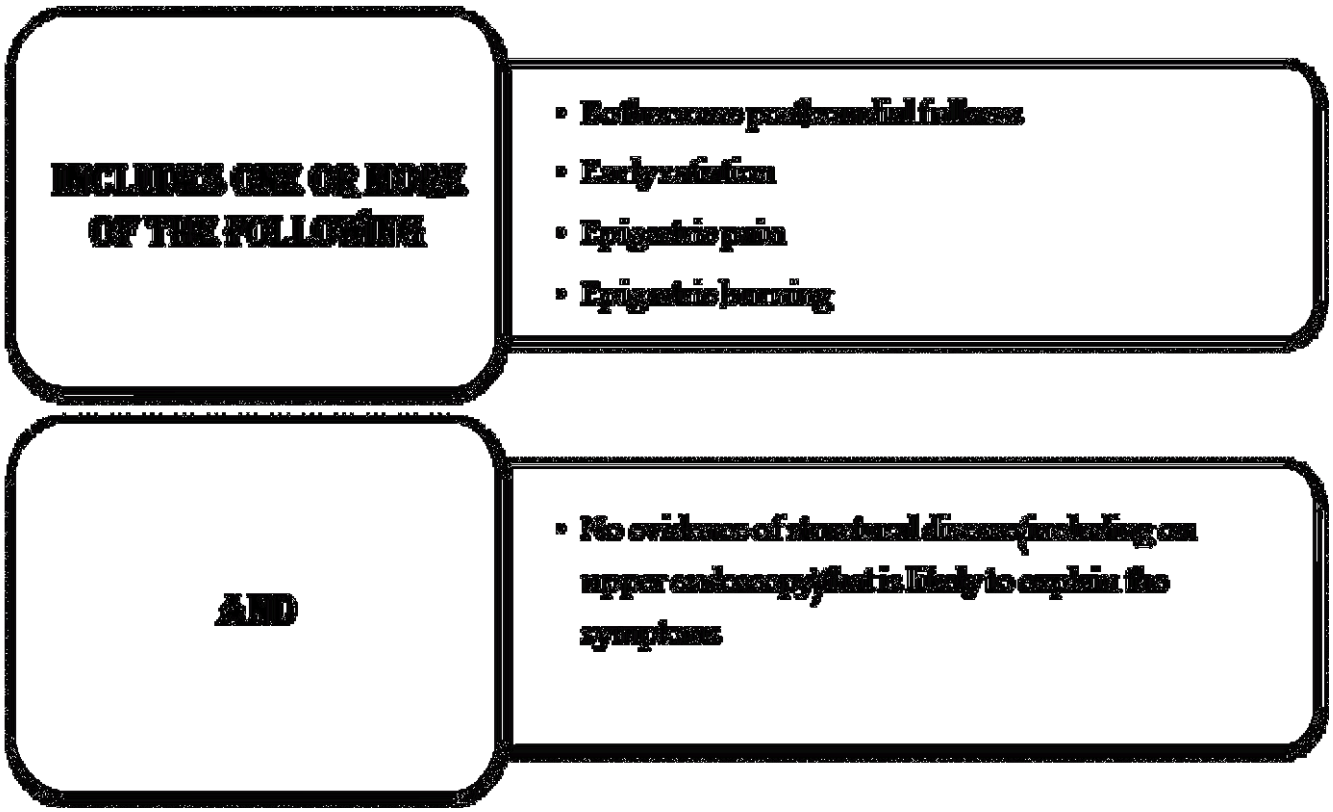
SUPPORTIVE CRITERIA

- **Pain may be of a burning quality,but without a retrosternal component**
- **Pain is commonly induced or relieved by ingestion of a meal,but may occur while fasting**
- **Postprandial distress syndrome may coexist**

POSTPRANDIAL PAIN SYNDROME



FUNCTIONAL DYSPEPSIA



ANATOMY

OESOPHAGUS

Oesophagus allows passage of food into the stomach and is 25-30 cm in length. Its origin is at the cricoid cartilage at the level of fifth cervical vertebra to oesophago gastric junction at the level of twelfth thoracic vertebra. It is located in front of spine and behind the trachea. It enters the thorax at the sternal notch and it passes

in the posterior mediastinum in the thoracic cavity. Oesophageal hiatus is opposite tenth thoracic vertebra.

Based on the anatomy,oesophagus is divided into

-cervical

-thoracic

-abdominal

Functionally it is divided into

-upper and lower oesophageal sphincter

-body of oesophagus

The pharyngo oesophageal junction is the narrowest part of alimentary canal. The superior,middle constrictor just above upper oesophageal sphincter help in deglutition and speech. Oesophagus has four constrictions

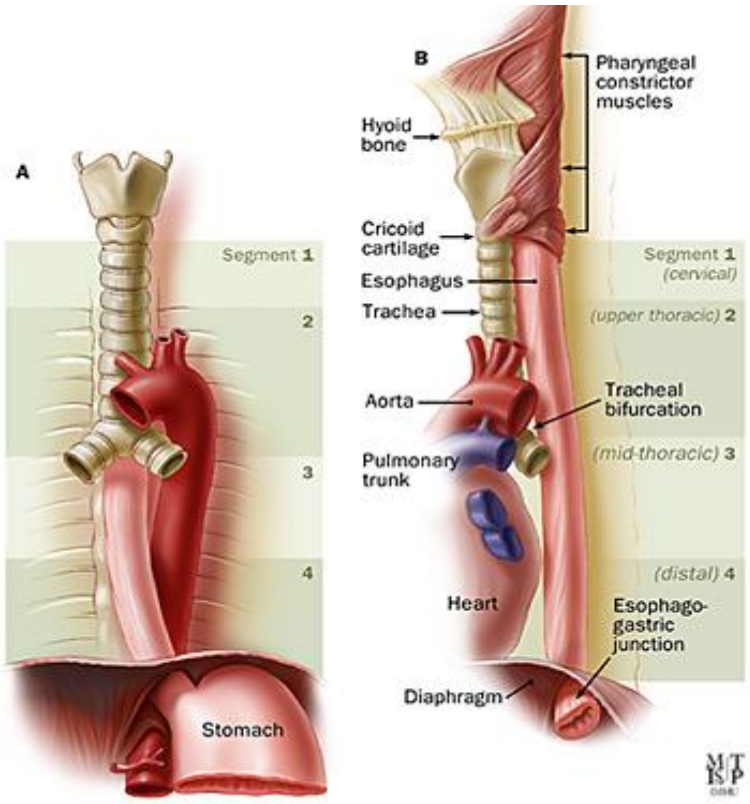
-at pharyngo oesophageal junction

-crossing of aortic arch

-crossing of left bronchus

. -when it pierces the diaphragm

The normal constriction should be kept in mind during endoscopy



UPPER OESOPHAGEAL SPHINCTER

The zone of high pressure at pharngo oesophageal junction is the upper oesophageal sphincter. Complex muscles of larynx and neck end at this level. The three pharyngeal constrictor muscles end at the crest of oesophagus.

The pharyngeal constrictor muscle along with posterior cricoarytenoid muscle anchor pharynx to structures of mouth. These muscles are responsible for swallowing and speech but they are not the reason for high pressure in upper oesophageal sphincter.

LOWER OESOPHAGEAL SPHINCTER

It is located at the oesophageal gastric junction allowing the food to enter the stomach.Lower oesophgeal sphincter is not a true sphincter. Atleast 2 cm length is required for normal functioning of sphincter. It has a high pressure zone of around 2-5 cm length with resting pressure of about 6 to 25 mm Hg. This transition can be measured using a manometric tracer called Respirator inversion point. Here the pressure changes to positive from negative during inspiration and vice versa during expiration.

Along with peristaltic contraction, relaxation of the lower oesophageal sphincter by vagal stimulation is required to open the sphincter. Vagal relaxation of sphincter lasts for 4 to 6 seconds and occurs 1.5 to 2.5 seconds after pharyngeal swallowing. This timed relaxation is required for food to enter the stomach from oesophagus. After the food had entered the stomach, lower oesophageal sphincter contracts and the pressure returns to the baseline and acts as a reflux barrier.

OESOPHAGEAL LAYERS

The two proper layers of oesophagus include mucosa and muscularis propria. Oesophagus does not contain serosal layer differentiating it from other gastro intestinal tract structures. The mucosa is made of squamous epithelium and is the innermost layer. The lowermost 1-2 cm is the transition zone where oesophageal

squamous epithelium and columnar epithelium of stomach meet at a point called as Z line.

The layers of oesophagus include

-epithelium

-basement membrane

-lamina propria

-submucosa

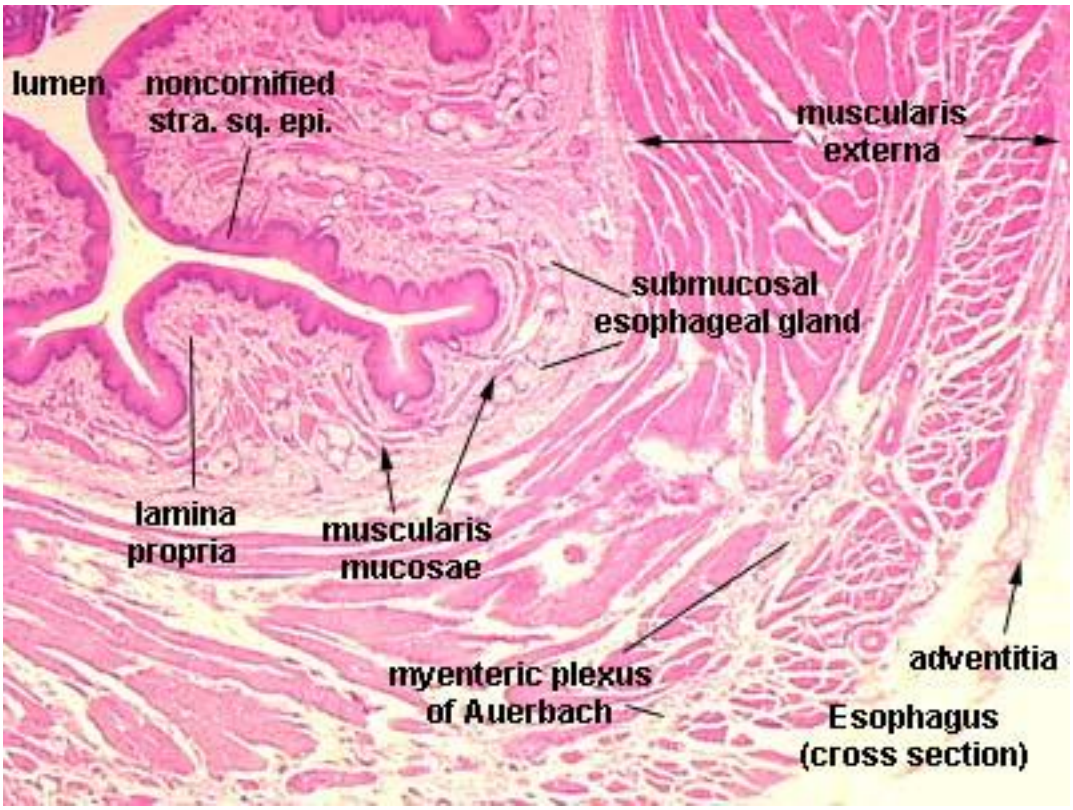
-muscularis mucosa

Histology

Outer to muscularis mucosa is submucosa and immediately outer to submucosa is muscularia propria. Oesophagus contains two layers of muscle bundle namely outer longitudinal and inner circular muscles. Upper one third of oesophagus has striated muscles and lower two third has smooth muscles.

The inner circular muscle is the extension of cricopharyngeus muscle and on reaching the stomach they continue as the circular muscles of stomach along the lesser curvature. This transition occurs at the collar of Helvetius at the level of cardiac notch. This layer of oesophagus contain the Auerbach's plexus of nerve

fibres. Surrounding these circular muscle layer is the longitudinal muscle layer. Similar to the circular muscle layer it extends into the stomach and alimentary tract.



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STOMACH

The stomach is the most dilatable and the widest part of alimentary tract.it is 25 cm in length. Stomach is divided into

-cardiac part

-pyloric part

Cardiac part is further subdivided into

-fundus

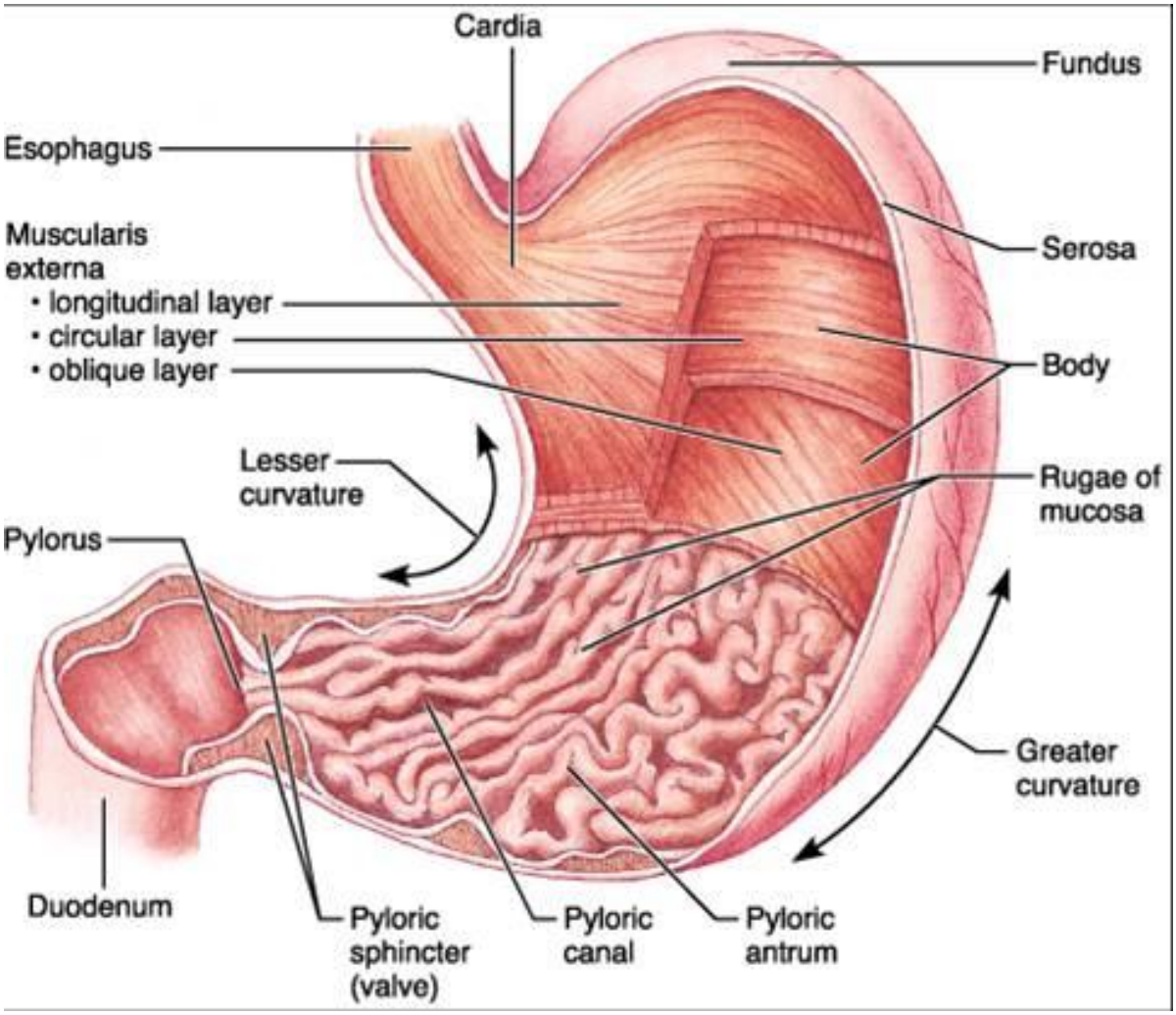
-body

Pylorus is subdivided into

-antrum

-canal

The part of the stomach which is situated above the cardiac end of stomach is the fundus of stomach. Body of stomach extends from fundus to incisura angularis along the lesser curvature. Pylorus is situated below the body of stomach which is divided into antrum and canal.



Layers of stomach are

-mucosa

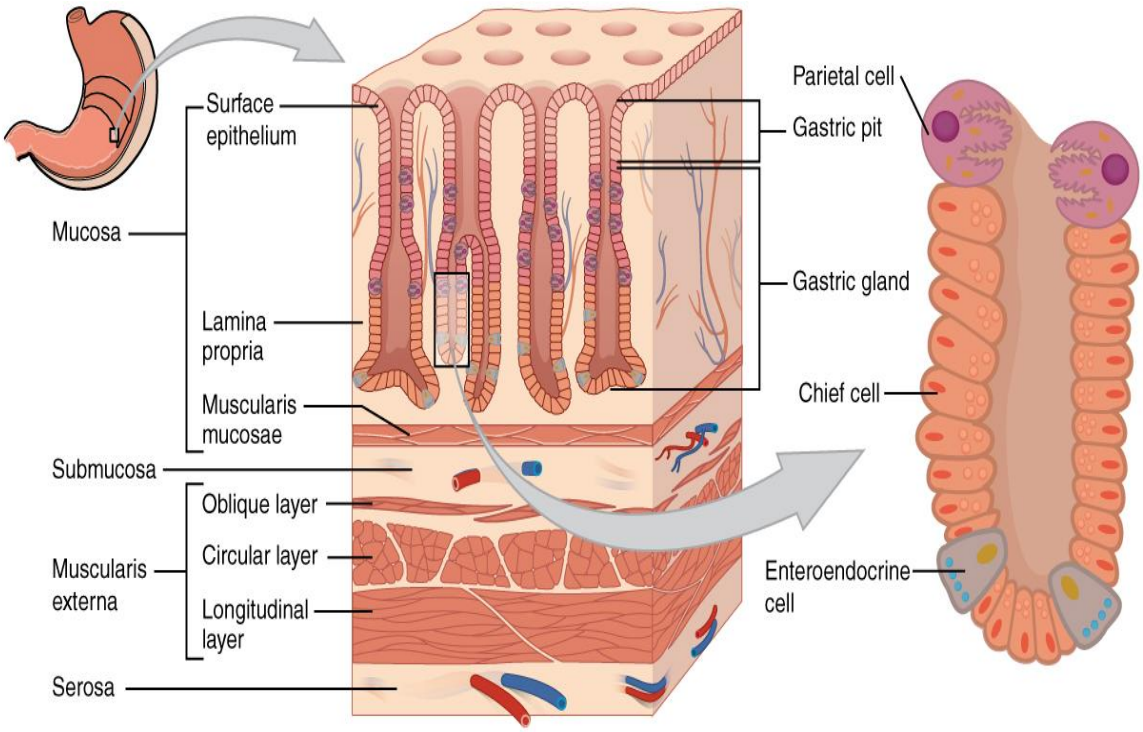
-submucosa

-muscle coat

-serosal coat

When the stomach is empty mucosa has rugae. These rugae are irregular except in lesser curvature where they are longitudinal. Rugae disappears in distended stomach. The single layer of lining epithelium is known as 'surface mucosal cells' as they secrete mucus. The mucus protects the stomach from action of enzymes and acids. Liquids usually reach the lesser curvature so it is the most common site of peptic ulcer. Mucosal layer contains many depression called gastric pits. Damage to the mucus layer directly exposes the stomach wall to the acids and enzymes and forms the basis for 'leaking roof' hypothesis of peptic ulcer.

Muscle layer has outer longitudinal and inner circular fibres. Circular fibres form the pyloric sphincter. Deepest layers namely oblique fibres form the gastric canal.



Gastric Glands

It can be subdivided into

-Cardiac glands

- Main gastric glands

- Pyloric glands

Cardiac glands

They are tubular glands located near the oesophageal opening and has secreting cells.

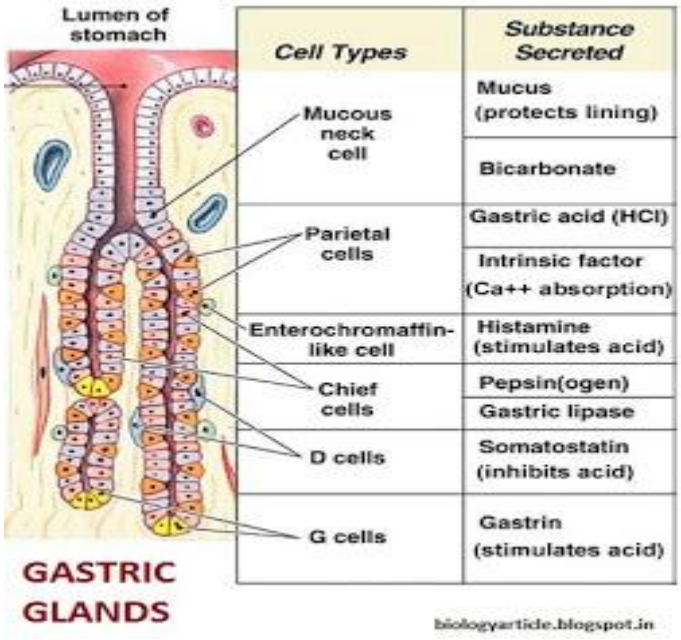
Main gastric glands

They are located in the fundus and body of stomach. They contain various cells namely peptic cells,oxyntic cells,neck cells and endocrine cells. Peptic cells secrete pepsin and located in the basal portion.

Oxyntic cells secrete hydrochloric acid and also intrinsic factor. Both oxyntic cells and mucous cells are located in the upper part. Endocrine cells namely D-cells and enterochromaffin like cells are present all over the gland secreting somatostatin and histamine respectively. Gastrin secreting cells are called as G-cells are located in the lower part of gastric glands. There are also many cells without any differentiation and their functions are not known.

Pyloric glands

They are located in the pylorus and secrete parietal,endocrine and mucous cells.



Cell Types	Substance Secreted
Mucous neck cell	Mucus (protects lining)
	Bicarbonate
Parietal cells	Gastric acid (HCl)
	Intrinsic factor (Ca++ absorption)
Enterochromaffin-like cell	Histamine (stimulates acid)
Chief cells	Pepsin(ogen)
	Gastric lipase
D cells	Somatostatin (inhibits acid)
G cells	Gastrin (stimulates acid)

PHYSIOLOGY

Around 2.5 litres of gastric juice is produced daily. Hydrochloric acid produced by the juice produce a medium for pepsin to act. The mucus content protects the gastric mucosa from acid by forming a gel like layer and also secretes bicarbonate so that though pH is less in the luminal side,pH becomes basic on the surface epithelial cells thus preventing the epithelial damage. Helicobacter pylori are

concentrated in the mucus layer and damage to the mucus layer leads to the pathogenesis of H.pylori.

Gastric secretion and motility are controlled by

a- neural mechanism by autonomic cholinergic neurons

b- hormonal mechanism by gastro intestinal hormones

The three phases of gastric secretions are

-cephalic phase

-gastric phase

-intestinal phase

Cephalic phase

By direct cholinergic stimulation through activation of vagal centre. Oxyntic cells and parietal cells are stimulated leading to digestive enzymes and acid secretion.

Gastric phase

Distension of stomach by food activates the vagal reflex which leads to the secretion of gastrin by the G-cells.

Intestinal phase

Similar to gastric phase, once the food enters the intestine ,neuronal receptors get activated leading to intestinal gastrin secretion. Once pH in the duodenum and antrum decreases, further secretion of acid is inhibited due to vagal inhibition.

STRUCTURAL CAUSES OF DYSPEPSIA

HIATUS HERNIA

It is herniation of part of the stomach through a defect or opening in the diaphragm. It occurs in 10% of the people but usually does not cause any symptoms[32].

Hiatus hernia leads to weakening of the lower oesoghageal sphincter leading to reflux which causes heart burn. But all cases of hiatus hernia do not cause heart burn.

GASTROESOPHAGEAL REFLUX DISEASE

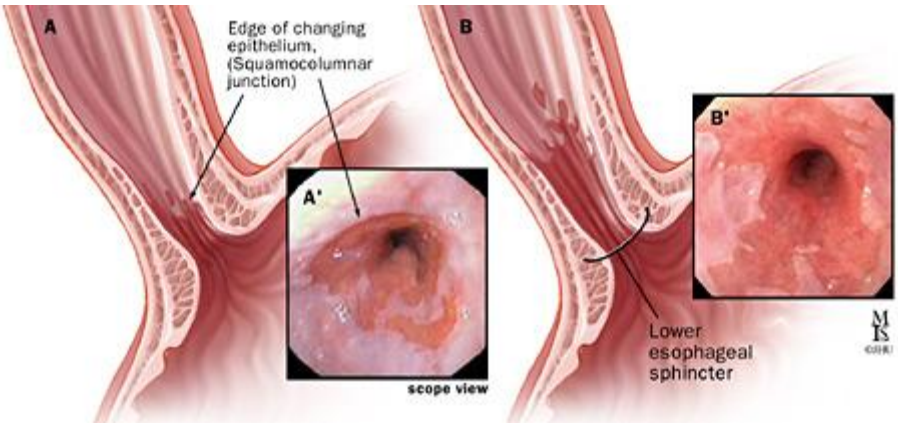
It occurs when the pressure in the lower oesophagus is low and hence does not prevent regurgitation of gastric contents or when the lower oesophageal sphincter undergoes spontaneous relaxation. Heart burn is the usual symptom. As said above hiatus hernia is one of the causes for regurgitation. Long standing heart burn is the usual symptom. It usually occurs in the epigastric area and doesnot radiate to the back.

BARRETT'S OESOPHAGUS

Long standing exposure to acid ultimately changes squamous epithelium to columnar epithelium [metaplasia] with goblet cells. It is a premalignant condition for development of adenocarcinoma. It is estimated that the risk of adenocarcinoma is 6-7 per 1000 patients[32]. Patients usually have heart burn,dysphagia,hematemesis and pain in retrosternal area. Barrett's oesophagus is not associated with severity of heart burn but associated with duration of heart burn.

Endoscopic biopsy helps in the diagnosis. But many patients donot have metaplasia. It is recommended that if two successive endoscopic biopsy is negative

then patient need not undergo endoscopy for 3 years. Low grade dysplasia can be managed conservatively but regular screening by endoscopic biopsy should be done. In case of high grade dysplasia,oesophagectomy, endoscopic mucosal resection or ablation can be done.



PEPTIC ULCER DISEASE

It is discontinuity of the mucosal lining of stomach or the first part of duodenum. It is due to the effect of pepsin and acid when the mucosal barrier get deranged. Acute ulcers may be shallow but chronic ulcers are deep and scirrhous.4% of gastric ulcer is due to malignancy.

Common sites of peptic ulcer are

- First part of duodenum
- Along the lesser curvature of stomach

- Prepyloric and pyloric channel

Gastric ulcer

- Usually seen in late middle age.
- incidence is same in both the sex and
- increases with age.

Duodenal ulcer

- common in middle age
- common in males in ratio of 3:1
- in 10 – 20% both gastric and duodenal ulcers will be present

Modified Johnson Classification of Peptic ulcer[33]

Type 1-ulcer along the body of stomach

Type2-duodenal ulcer along type-1

Type3-ulcer within 3 cm of pylorus

Type4-ulcer in the upper part of stomach

Type4-ulcer can occur anywhere in the stomach.

Type 5-with NSAID usage

Type2 and 3 are associated with excess acid secretion.

Causes

-H pylori is the most common cause

-NSAID,aspirin

-smoking

-spicy and irregular diet, coffee

-endocrine causes like Zollinger Ellison syndrome, parathyroidism

-emotional factors

-genetic

Pathogenesis

-loss of mucosal defence

-excessive acid secretion mainly in duodenal ulcer

-decreased bicarbonate secretion

-decreased prostaglandin secretion

H pylori hydrolyses urea to produce ammonia which in turn increases gastrin secretion.

Symptoms usually include abdominal pain,bloating,nausea,loss of weight and melena.

Diagnostic tests include barium study,endoscopy and endoscopy with biopsy.

The various tests for H pylori include

-urea breath test

-culture of the biopsy specimen

-rapid urease test

-stool antigen test

-histological examination

Treatment

-reassurance

-medical treatment

a.H2 blockers

b.proton pump inhibitors

c.antacids

d.prostaglandin analogue

e.prokinetics

HELICOBACTER PYLORI

The discovery of H pylori and its relation with gastritis was discovered in 1982 by Warren and Marshall led to the various studies regarding the pathogenesis of gastro duodenal lesion.

It is a gram negative bacteria which is spiral shaped and motile and it colonises in the submucosal layer over the epithelial cells. Though it can be seen throughout the stomach it is mainly concentrated in the antrum of thestomach, as parietal cells are less here.

Faeco-oral or oro-oral route are the path of transmission. So H pylori are demonstrated in gastric juice and saliva by PCR method.

It is more common in developing countries like India. Poor living condition is attributed to its high prevalence in developing countries. They are equally present in both males and females and rate is high among smokers.

PATHOGENESIS OF H. PYLORI INFECTION

Cag A gene is said to be present in most of the patients with gastric ulcer or carcinoma of stomach.H pylori contains Cag A gene but toxigenic in only 40% strains. It reaches the mucosa through its characteristic motility and adheres to the mucosa. The organism contains urease which produces a basic environment by converting urea to ammonia and it protects itself from the gastric acidity.

As the organism is located to the mucosal lining the various immune cells cannot reach there, thus protecting the organism. But the vicious cytotoxic immune response leads to the damage to the gastric mucosa leading to ulcerative lesion. So it is the immune response to the organism which produces the ulcer and not directly the organism. Thus, though the organism is non invasive, it produces gastritis indirectly by inflammatory response. If the infection gets established it stimulates the G- cells to release gastrin leading to hypergastrinemia. Hypergastrenemia in turn lead to duodenal ulcer by stimulating parietal cells to produce acid.

But the exact role of H pylori in pathogenesis of gastric ulcer is not known.it is thought to be due to reduced gastric resistance to pepsin and acid[34].

The host immune response is similar to other bacterial infection- acute neutrophilic infiltrate followed by chronic cell infiltrate with Th1 response predominating.

In association with H pylori chronic gastritis is classified[35] into

Type A- atrophic with parietal cell antibodies

Type B-no parietal cell antibodies

Type AB-both atrophic and patchy

Chronic gastritis has the risk of development of adeno carcinoma.H pylori infection is more strongly associated with duodenal ulcer than gastric ulcer with prevalence almost 100%.

Diagnosis of H pylori infection

There are invasive and non invasive method

Invasive

-culture

-histopathology of the biopsied specimen

-rapid urease test

Non invasive

-serology

-urea breath test

- stool antigen test

-PCR

-Urine antigen

The choice of the appropriate test depends upon

-sensitivity and specificity

-cost

-availability

-patient willing to undergo endoscopy

The main use of history taking and examination is to

-to find if dyspepsia is due to NSAID induced or due to GERD

-If the patient has alarm symptoms or not

Patients with reflux symptoms should be treated as GERD.Patient with alarm symptoms should be investigated early, and endoscopy should be done. Though malignancy is rare,it is more common in patients with alarm symptoms than with simple dyspepsia.

MANAGEMENT OF DYSPEPSIA

Dyspepsia can be managed usually in five different methods[35]

a.acid suppression

b.H pylori non invasive test as mentioned above followed by H pylori eradication therapy for positive cases

c.non invasive test followed by endoscopy for positive cases

d.empirical H pylori treatment without non invasive or invasive tests

e.early endoscopy

With alarm symptoms

Patients with alarm symptoms after age of 55 years should have early endoscopy done as there is clear cut increase in malignancy rate. The optimal age ischosen as 55 years because below which the incidence is less than 10 per 100,000.

Without alarm symptoms

In treating patients with new onset dyspepsia without any alarm symptoms in majority of cases empirical acid suppression drugs are usually prescribed initially. However as mentioned above there are other options like testing for H pylori and starting eradication therapy in positive cases or doing endoscopy in positive cases

As the incidence of ulcer is low in new onset dyspeptic patients below 45 years American college of physicians based on various studies recommended acid suppression antisecretory therapy as the treatment of choice below 45 years of age without organic disease. They also suggested that if the symptoms do not decrease in 7- 10 days or if the symptoms have not completely resolved in 6-8 weeks, then endoscopy should be done. But this age threshold is still debatable.

Collected data suggest that initial H pylori test followed by treatment is more cost effective than empirical acid suppression therapy.

Various therapies of treatment include

1.PPI therapy

2.Acid suppression

3.Prokinetic therapy

4.H.pylori eradication therapy

5.Psychological therapy

Among these therapies, only PPI therapy and H.Pylori eradication therapy are highly efficacious in treating functional dyspepsia[36]. In patients who are H.Pylori positive, H pylori eradication therapy is very cost effective as it has a long term effect when given once. In patients who don't respond to eradication

therapy and who are H pylori negative, PPI therapy for one month is recommended.

Many studies have tried to establish association between H pylori and non ulcer dyspepsia so that the patients can be treated by eradication therapy.But no concrete evidence is available.In Germany ,a study done was by Bajorsky et al, he came to a conclusion that H pylori infection per se causes dyspepsia[37]. In that study when all important causes of dyspepsia was excluded,85% HP-positive patients showed improvement after eradication therapy.But in this,in many patients the symptoms decreased partially only,they did not recover fully which proves that inflammatory infiltrates partly persists in gastric mucosa.This proves that HP gastritis is an important etiological factor in causing dyspeptic symptoms.

In 1997,the first national workshop on H pylori was held in Mumbai ,which had these significant conclusions[38]

- H pylori infection in healthy and asymptomatic persons- prevalence rate in India varies from 31 to 84%
- Factors like socioeconomic status,age,sanitation,housing and methods used in diagnosis mainly determines the prevalence rate
- In India the age related prevalence study showed that infection occurs at an earlier age here compared to western countries

- No association was found between degree of gastric inflammation and non ulcer dyspeptic symptoms and the frequency of non ulcer dyspepsia varied from 60-85%
- There is no big study contributing the association between histological picture of gastric mucosa and its symptom response
- The preferred mode for diagnosis of H pylori infection is invasive techniques and among them rapid urease test is very popular.

The house urease test was found to be cheap and more sensitive when compared to Helicochek kit, the commercially available one.This was done by Anitha Kamath et al, using histology as gold standard she compared the sensitivity and specificity of both the tests.

The prevalence of H pylori infection in gastroduodenal diseases was studied by Thayumanavan L et al a Madurai[38]during routine upper gastrointestinal endoscopies.He came to a conclusion that in southern parts of Tamil Nadu the organism has a wider prevalence rate and also for detecting the organism rapid urease test is cheap,useful and simple and its prevalence is almost as high as non ulcer dyspeptics as those with duodenal ulcer.

Also serology is ideal for epidemiological studies.For confirming eradication in research setting, culture should be included in the protocol till urea breath test is available widely.

The study by Warren Marshall concluded that more than 90% of patients with duodenal ulcer was infected with H pylori when it was compared with 40% of control group. In 43-79% of NUD patients H pylori infection was evaluated by four endoscopic surveys.These numbers were above the control group in 3 of these surveys conducted.Also no symptoms was seen in 50% of infected persons.Reflux like or ulcer like symptoms were seen in these infected persons.

A statistically significant and convincing association was established by EUROGAST study group[40] . It showed a positive association between H pylori and gastric cancer in different parts of the world.

In patients with upper GI symptoms using the rapid urease test a significant prevalence was seen in both ulcer and non ulcer dyspepsia by PC Jain et al.

Despite normal endoscopy ,significant mucosal lesions were seen in patients with infection by histopathological evidence. His study was done in Nigeria. Otherwise there are few studies on histopathological evidence and its relation with the organism. In India there are no studies.

H.PYLORI ERADICATION THERAPY

Whether the patients are suffering from initial presentation of the disease or its recurrence, if they have gastricor duodenal ulcer and if they are infected with H pylori, they should be treated with antimicrobials. Any drugs that is known to cause dyspepsia should be stopped or substituted if possible. But it remains controversial in treating H pylori in non ulcer dyspepsia patients. A negative test for H pylori atleast 28 days after therapy is considered as H pylori eradication.

FACTORS WHICH MODIFY TREATMENT

- Urease based tests may fail to detect residual infection or recurrence if treated priorly with PPI or bismuth as they inhibit urease enzyme.
- Biopsy based tests become inaccurate because H pylori tends to move to proximal part of stomach during suppression of acid seceretion.
- The gold standard test to confirm eradication is C^{13} and C^{14} urea breath test as they sample the whole stomach.It takes atleast 6 months for antibody titre to fall significantly so serology is no useful to confirm eradication
- In 50-80%, dual therapy with a 2 week combination of ranitidine or omeprazole or bismuth citrate and either clarithromycin or amoxicillin eradicated H pylori. Eradication rate may be 50-70% in triple therapy.PPI

based therapy for 1wk twice daily caused eradication in about 90% patients.7days treatment with omeprazole and amoxicillin and metronidazole or a PPI regimen is considered second line.

TRIPLE REGIMENS WITH AMOXCILLIN AND METRONIDAZOLE

	REGIMEN 1	REGIMEN 2	REGIMEN 3
Drug	**Omeprazole+ Amoxicillin+ Metronidazole**	**Ranitidine+ Amoxicillin+ Metronidazole**	**Bismuth+teracycline/ Amoxcillin Metronidazole**
Dose (daily)	**40mg once+ 500mg thrice+ 400mg thrice**	**300mg once+ 750mg thrice 500mg thrice**	**120mg 4 times+ 500mg 4 times+ 200-400mg 4 times**
Duration	**7 days**	**12 days**	**2 weeks**
Efficacy	**95%**	**90%**	**60-90%**
Side effects	**Diarrhea,nausea with metronidazole**		

The above triple regimens are now been replaced by amoxicillin or clarithromycin along with a PPI which are shorter regimens. In eradication they are equally effective.

LOW DOSE TRIPLE THERAPY

LOW DOSE TRIPLE THERAPY

	REGIMEN 1	REGIMEN 2
Drugs	**PPI+Clarithromycin+ Metronidazole**	**PPI+Amoxicillin+ Clarithromycin**
Dose(daily)	**Once/twice daily+ 250mg twice+ 400mg twice**	**Twice+ 1gm twice+ 250-500mg twice**
Duration	**7 days**	
Efficacy	**90%**	**90%**
Side effects	**Uncommon:diarrhea,nausea with metronidazole**	

QUADRAPLE THERAPY

PPI(once/twice daily)+

Colloid bismuth sub citrate(120mg 4 times daily)+

Tetracycline (500mg 4 times daily)+

Metronidazole(400-500mg 3-4 times daily)

Duration of therapy: 7 days

Efficacy : 85-95%

Side effects:diarrhoea,nausea

SEQUENTIAL THERAPY

DAY 1-5

PPIs twice a day

Amoxicillin 1 gm twice a day

DAY 6-10

PPIs twice a day

Clarithromycin 500 mg twice a day

Tinidazole 500 mg twice a day.

98% eradication rate is obtained by sequential therapy. It is suggested that sequential therapy should be made as the standard eradication therapy for H pylori as it gives a higher eradication rate than the conventional triple therapy.

SURGICAL TREATMENT

a.Anti-reflux surgery like fundoplication

b. Endoscopic therapy

Recently developed therapies likeradiofrequency energy and Plexiglas injection are less invasive than anti-reflux surgery like fundoplication.

c.Peptic ulcer surgery

1.Truncal vagotomy

Division of the main trunk of the vagus resulting in denervation of pylorus part of the stomach. In case of truncal vagotomy pylorus drainage procedure is needed.

2. Selective vagotomy

Only the nerve of Latarjet which arises below the celiac and hepatic branches is divided. This procedure also requires pylorus drainage procedures This procedure is usually not done.

3.Highly Selective Vagotomy

It involves only the division of branches innervating the antrum and pylorus of stomach. Drainage procedure is not needed as the pyloric part is not denervated.

In some cases vagotomy with anterectomy is done like

Bilroth I

Roux-en-Y Gastrojejunostomy

These procedures have the low recurrence rate.

GASTRIC CANCER

It is the fifth leading cause of cancer in the world and third leading among death due to cancer[41]. It is common in East Asia and East European countries. In India it is more common in southern india. Incidence of gastric cancer in India in 2001 was 35,675 (23,785 males and 11890 females)[42].

Etiology

Exact etiology is not known. Various risk factors are

a.H pylori –though 65-80% gastric cancer patients are H pylori positive only 2% of infected persons develop malignancy.it is due to long standing inflammation or virulence factor like Cag A[43]

b. Smoking is an important risk factor.

c.Dietary factors-smoked food, processed meat, pickled vegetables, salt rich food

d. Other risk factors include iodine deficiency, genetics, immune compromised state,chronic atrophic gastritis,intestinal metaplasia, genetic.

Symptoms

-often asymptomatic

-non specific symptoms like abdominal pain ,discomfort, bloating, nausea, vomiting, weight loss, haemetemesis, melena, loss of weight and appetite, weakness, fatigue

Investigation include endoscopy followed by endoscopic biopsy of any suspected lesion in stomach, CT abdomen and pelvis.

Treatment

Surgery is the treatment of choice for gastric cancer.it includes

a.endoscopic mucosal or sub mucosal resection

b.wedge resection

c.partial or subtotal gastrectomy

d.laproscopic gastrectomy

FUNCTIONAL DYSPEPSIA

When the chronic or recurrent gastrointestinal symptoms cannot be explained by structural or biochemical abnormalities it is called as functional dyspepsia. It can be due to dysfunction right from oropharynx to large intestine including the biliary tract.

It is a heterogenous syndrome. They can be subdivided which may suggest the possible underlying pathology.

It includes

Ulcer like dyspepsia

- they usually have the typical symptoms of peptic ulcer

Dysmotility-dyspepsia

- symptoms that suggest gastric stasis or intestinal dysmotility like nausea, fullness and regurgitation

Reflux-like dyspepsia

- epigastric pain with heart burn or regurgitation

H pylori has not been associated significantly with non ulcer dyspepsia in epidemiological studies. Various other factors like increased acid secretion, stress, dietary factors and psychological factors have not been clearly or convincingly associated with non ulcer dyspepsia.

Pathophysiology

a. Delayed gastric emptying

It occurs when the antral peristalsis gets impaired or when the duodenal resistance is increased. The measurement of gastric emptying in relation to a meal is used to assess the efficacy of gastric neuromuscular work. It usually ranges between 20-50%. Few patients have delayed gastric emptying for solids. Studies have shown that it is more common in females than males. But many studies have shown that there is no relation between delayed gastric emptying and dyspepsia.

Postprandial intake of water decreases the antral gastric motility and increase in cholecystokinin. This is due to flow of fatty chyme into the duodenum which in turn inhibits antral peristalsis known as ‘duodenal break’. Similarly low viscosity meal delays gastric emptying more compared to high viscosity meal because of the rapid initial inflow into the duodenum which inhibits the antral peristalsis. So these may be more important factors in causing symptoms especially in postprandial distress syndrome.

b.Hypersensitivity to gastric distension

Some patients with functional dyspepsia are hypersensitive to gastric distension. It is related to

-mechanoreceptors or chemoreceptor sensitization

-increased exitability of spinal cord neurons

-dysfunction of spinal inhibitory symptoms

-altered CNS modulation and processing of visceral stimuli

c. Altered duodenal sensitivity to lipids

Nutrient lipids increases the perception of gastric distension as it releases cholecystokinin. Patient with functional dyspepsia had increased duodenal acid

exposure compared to normal people on duodenal pH monitoring. This is due to impared acid clearance.

d. Impared gastric accommodation

Proximal stomach functions as a reservoir of food following a meal while the distal stomach is responsible for grinding the particles small enough for emptying into the pylorus. If relaxation of the proximal stomach through vagal reflux is impaired then large volume of food cannot be accommodated in the proximal stomach. Various studies have shown that impaired gastric accommodation is seen in around 40% of functional dyspeptic patients.

Various other mechanism leading to dyspepsia include

- unsuppressed contraction of proximal stomach in the postprandial phase
- myoelectric abnormalities

OTHER CAUSES

-metabolic disturbance

-biliary disease

-irritable bowel syndrome

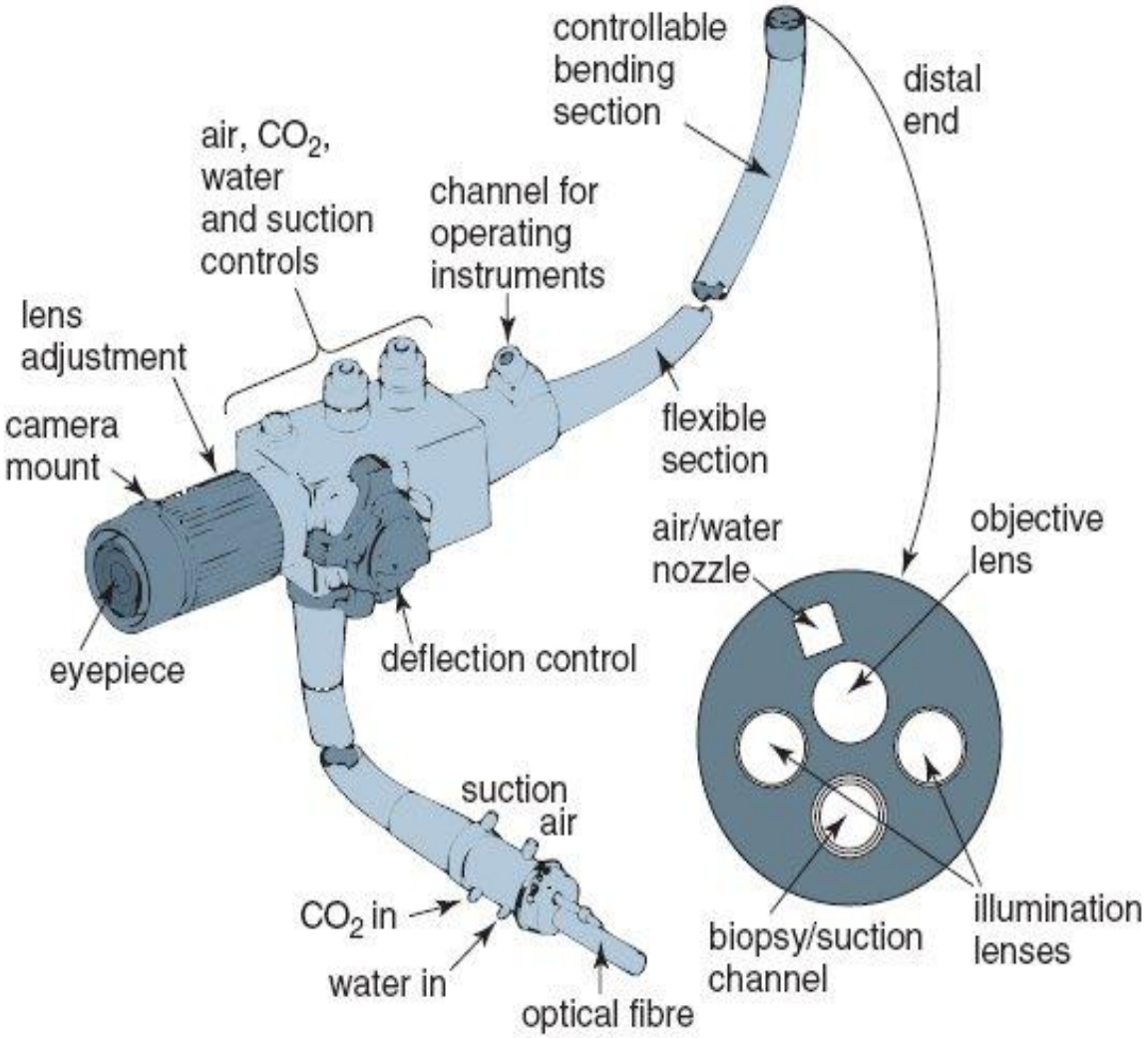
-pancreatic disease

-psychiatric disease

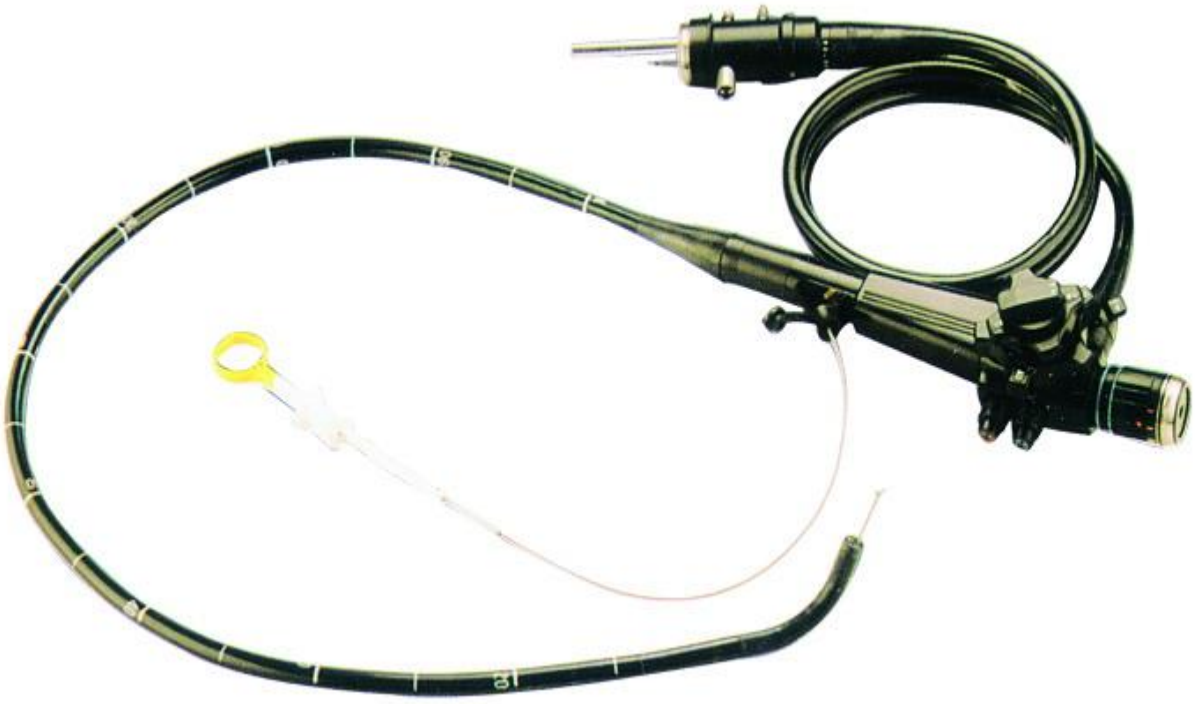
ENDOSCOPY

HISTORY AND DEVELOPMENT

Earlier rigid endoscope with smaller lamps were used. But it had the disadvantage of overheating and also due to its rigidity it cannot be passed through the curvatures of intestine. It was initially designed by Polish surgeon Johann Von Mikulicz Radecki of 19th century.



Flexible endoscopy(semi rigid) was first introduced by R.Schindler in 1936in association with Georg Wolf a German physician. Flexible fibroscope was first discovered by Baird in 1928.



The first clinically usable flexible endoscope was discovered by C.Wilbur Peters in association with Lawrence Curtis, a physics student. The problem of fibre cross talk which made the interpretation impossible was rectified by the discovery of glass coating of fibres for insulation. This lead to the development of fiberscope.

The controllable tip gastroscope was discovered in 1962 by Hisschowitz . The first commercial endoscope was made by Inc Norwalk CT in 1961. Various studies

were done to look for the usefulness against the complication of endoscope and today it had developed to an extent that endoscopic surgeries are being done.

INSTRUMENTATION

Various flexible endoscopes are available based on their dimensions, direct viewing or video. The primary endoscope is a zero degree forward viewing. In case of duodenoscope it is 90 degree. Endoscope have a control head and an eye piece. The shaft has deflection ranging from 90-240 degrees in vertical position and 100 degrees in horizontal direction.The insertion tube has diameter ranging from 5.5 mm to 11 mm.

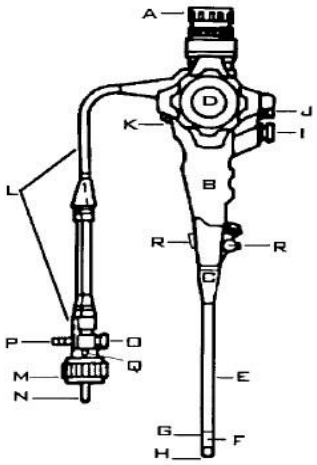
The deflection tip is controlled by 2 knobs located on the control head. The larger knob controls up and down deflection and a smaller knob controls the horizontal deflection. In addition to the knobs there are two buttons.the top button when pressed produces suction and the lower button produces air insuffulation. The lower button when pressed releases water for cleaning the tip.In case of video endoscope there are buttons which are used to take images.

Introduction:

Knowing your scopes.

1-External Parts:

A. Ocular/Eyepiece
B. Body Covers
C. Body Cone
D. Control Section
E. Insertion Tube
F. Bending Section
G. Bending Rubber
H. Distal End
I. A/W Buttons Valves
J. Suction Valve
K. CO2
L. Light Guide Section
M. Connector
N. Probe/Light Guide
O. Water Bottle Connector
P. Suction Port
Q. Ground Lug
R. Sub Water Feed



2. Internal Parts:

A. Image Guide Bundle
B. Light Guide Bundle
C. Forcep Channel
D. Air Line
E. Water Line
F. Drum Cable & Stoppers

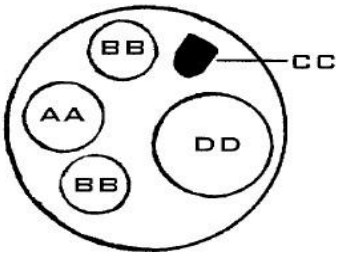
H = DISTAL END ASSEMBLY

AA = IMAGE GUIDE LENS

BB = LIGHT GUIDE LENS

CC = AIR WATER NOZZLE

DD = OPENING OF BIOPSY CHANNEL



The shaft in the flexible endoscope is 110-120 cm in length. The length of the working channel varies between 2 mm to 3.7 mm. In case of duodenoscope it varies between 3.2 to 4.2 mm.

Biopsy forceps, brush to take cytology specimen and other instruments can be passed through accessory channel. At present a double lumenendoscope is also available for usage.

The light source usually 300w xenon or halogen tungsten lamp is connected to endoscope. Also the water and air pumps are attached to the endoscope for suction and insufflation. Video monitor if needed is connected to the endoscope.

Good positioning and manipulation of endoscope is needed for efficient examination. Usually left hand is used to hold the control head, thumb holds the knob and middle and index finger hold the air insufflation and the suction buttons. The right hand adjusts the flexible shaft during examination.

Endoscopy is the investigation of choice for patients with upper gastrointestinal problems. So clinician specialised in uppergastrointestinal tract should be well versed in endoscopy.

PATIENT PREPARATION

First the procedure is explained to the patient in an understandable way and their consent is obtained. Previous treatment history is reviewed. The need for any medication like antibiotics are assessed. Patient should be advised overnight fasting. As most of the cases are out patients an attender should accompany them.

In case of relaxed patients, sedation may not be required but anxious patients can be sedated. A lignocaine spray for topical application can be tried before sedation. Sedation is usually done by diazepam or midazolam.

TECHNIQUE

INTRODUCTION OF ENDOSCOPE

Patient is positioned in left lateral decubitus position after the required topical anaesthesia or sedation is given. A mouth piece is placed between the teeth. Endoscopy is advanced along the midline and structures in the oral cavity are visualised. Once the endoscope reaches the cricopharyngeus, patient is asked to swallow and gentle pressure is given so that endoscope enter the oesophagus.

Examination of oesophagus

For good vision optimal insufflation is needed and the endoscope tip should be in the centre of the lumen. If the vision is obscured scope should be withdrawn.

As the endoscope passes through the lumen of the oesophagus, external compression at the four points asmentioned above will be noted. Oesophago gastric junction is usually noted at 38-40 cm from incisors and also there will be colour difference of the mucosa known as Z lines. To identify the position of oesophageal hiatus, patient is asked to inhale deeply so that diaphragmatic hiatus creates an imprint on the wall of oesophagus and stomach.

Passage into the stomach

Before entering the stomach the lower oesophageal junction should be looked for - whether it is normal, lax or constricted. Since the lumen of stomach is large the endoscope enters without any resistence. Patient may observe discomfort as the stomach gets distended with air. Greater curvature and the posterior wall is visualised once the tip is slightly turned downwards to the left. All the liquids in the stomach is suctioned for clear view of the stomach and also to prevent aspiration. If the endoscope is rotated the entire body of the stomach can be visualised. The proximal part of the curvature is visualised using J manoeuvre.J manoeuvre is done by rotating the scope by 180 degree upward when the stomach is distended.

To view the antrum, tip of endoscope is angulated and the pyloric ring is observed directly. The endoscope is passed through the pylorus under direct vision. Once passed through the pylorus duodenal first part can be visualised till the superior duodenal angle.

After viewing the duodenum endoscope is pulled out with stomach in the distended position to assess the proximal part of stomach along lesser curvature. When the endoscope is pulled back through the oesophagus the tip should be straightened.

Once the procedure is over, patient is observed and should avoid taking food or liquids for next 30 min.

ENDOSCOPIC BIOPSY

It is usually done once if there is any suspected lesion in the esophagus, stomach or duodenum. This include strictures, ulcers, mass lesions, polyps etc. Usually multiple specimens are taken to increase the sensitivity. If malignancy is suspected six biopsy specimen are usually taken. This will have sensitivity of more than 90-95%. If any bleeding varices is suspected biopsy should not be taken as this may lead to bleeding.

Biopsy forceps are available in various dimensions. Biopsy instrument is introduced into the working channel, the jaws of the instrument opened and pressed on the mucosa and then closed and pulled back. If the forceps contain a spike, multiple biopsies can be taken without removing the forceps from the working channel for each biopsy specimen.

Biopsy for stomach ulcer are usually taken at the base, that is the transition between the normal and abnormal mucosa in all four quadrants to improve the diagnostic accuracy. In case of mass in submucosal layer it is quite difficult to reach. Several biopsies need to be taken but there is always the risk of iatrogenic perforation.

Polyps should be biopsied to rule out malignancy. For this purpose hot or cold forceps can be used for small polyp less than 5 mm and snare is used for large polyps or pedunculated polyp. Specimen should be obtained in case of oesophageal stricture.

Japanese investigators have developed a suction apparatus which grasps the lesion and then the snare is placed at the base and lesion can be removed easily. If bleeding occurs coagulation technique is used.

Advantages of fibroscope[44]

a. there are no blind spots, i.e., there is no part which cannot be visualised.

b. fibroscope has made the procedure more simplified

c.direct vision for biopsy has revolutionised the biopsy procedure

d.recording ability has been improved.

e.supplemental techniques are available like washing and application of pigment solution.

Over all, the diagnostic accuracy has improved with advent of fibroscope.

METHODOLOGY

This is a prospective clinical study done at Kilpauk Medical College,Chennai, to find out the role of endoscopy in dyspeptic patients. The study duration was ten months from October 2013 to July 2014. Patients were selected using convenient sampling.

Patients were included after getting their informed written consent for the procedure. Thorough clinical history was obtained through the proforma including the presence of alarm symptoms. Basic investigations were done. Ultra sonogram

of the abdomen done to look for gall stone or any mass lesion. Then the patients were subjected to endoscopy and findings were noted.

All the instruments were washed with distilled water to prevent error due to pH change.

Inclusion Criteria

a.patient age should be above 12 years

b.patient for whom endoscopy was not previously done.

Exclusion criteria

a.patients below 12 years

b.already known case of liver disease, pancreatitis,gall stone

c.patient with unstable cardiac status

d.pregnant women

e.patient for whom endoscopy has already been done

f.intake of NSAID, antibiotics or proton pump inhibitors within 15 days

g.gastro intestinal bleeding

h.severe systemic illness

i.patient not willing for the procedure

PROCEDURE

All the patients were on out patient basis. Cardiology fitness was obtained for all the patients above 40 years before the procedure[47]. Patient were on fasting for 12 hours before the procedure.

For most of the patients endoscopy was done under topical anaesthesia itself. Only few patients required sedation. Lignocaine spray was applied 10 minutes before the procedure. Diazepam 5-10 mg i.v was given for sedation. Patient was put in left decubitus position.

The upper oesophago gastroduodenoscopy was done using Fujinon fibreoptic endoscopy.

Under direct vision endoscope was introduced with the tip in the centre of the lumen. Under optimal insufflation oesophagus is looked for inflammation, ulcer, growth and stricture. As the endoscope passes through the lumen of the oesophagus external compression at the four points as mentioned above will be noted.

Oesophago gastric junction is usually noted at 38-40 cm from incisor and also there will be colour difference of the mucosa known as Z lines. To identify the position of oesophageal hiatus patient is asked to inhale deeply so that diaphragmatic hiatus creates an imprint on the wall of oesophagus and stomach.

Before entering the stomach the lower oesophageal junction should be looked for whether it is normal, lax or constricted. Since the lumen of stomach is large the endoscope enters without any resistence. Patient may observe discomfort as the stomach gets distended with air. Greater curvature and the posterior wall is visualised once the once the tip is slightly turned downwards to the left. All the liquids in the stomach is suctioned for clear view of the stomach and also to prevent aspiration. If the endoscope is rotated the entire body of the stomach can be visualised. The proximal part of the curvature is visualised using J manoeuvre.J manoeuvre is done by rotating the scope by 180 degree upward when the stomach is distended.

To view the antrum tip of endoscope is angulated and the pyloric ring is observed directly. The endoscope is passed through the pylorus under direct vision. Endoscope can be advanced upto the second part of duodenum.

Endoscopic biopsies were taken randomly in case of normal study. In case of ulcers, six specimens were taken, four from the edge of the ulcer and two from the antrum of the stomach.

Among the six, two specimens- one from lesion site and one from the antral site and were inoculated in urea broth.It contains 0.5 ml of 1% Urea in distilled water.

If H pylori is present the colour of the solution will change from yellow to pink due to phenol red which acst as the indicator.

Other four specimens are sent in formalin for histopathological examination. Specimen are fixed with 10% formalin, then processed with paraffin and sections are cut. Two specimens are stained with haematoxylin-eosin stain and other two with Giemsa stain.

The H pylori test was considered positive if rapid urease tested positive or when histopathological examination was positive.

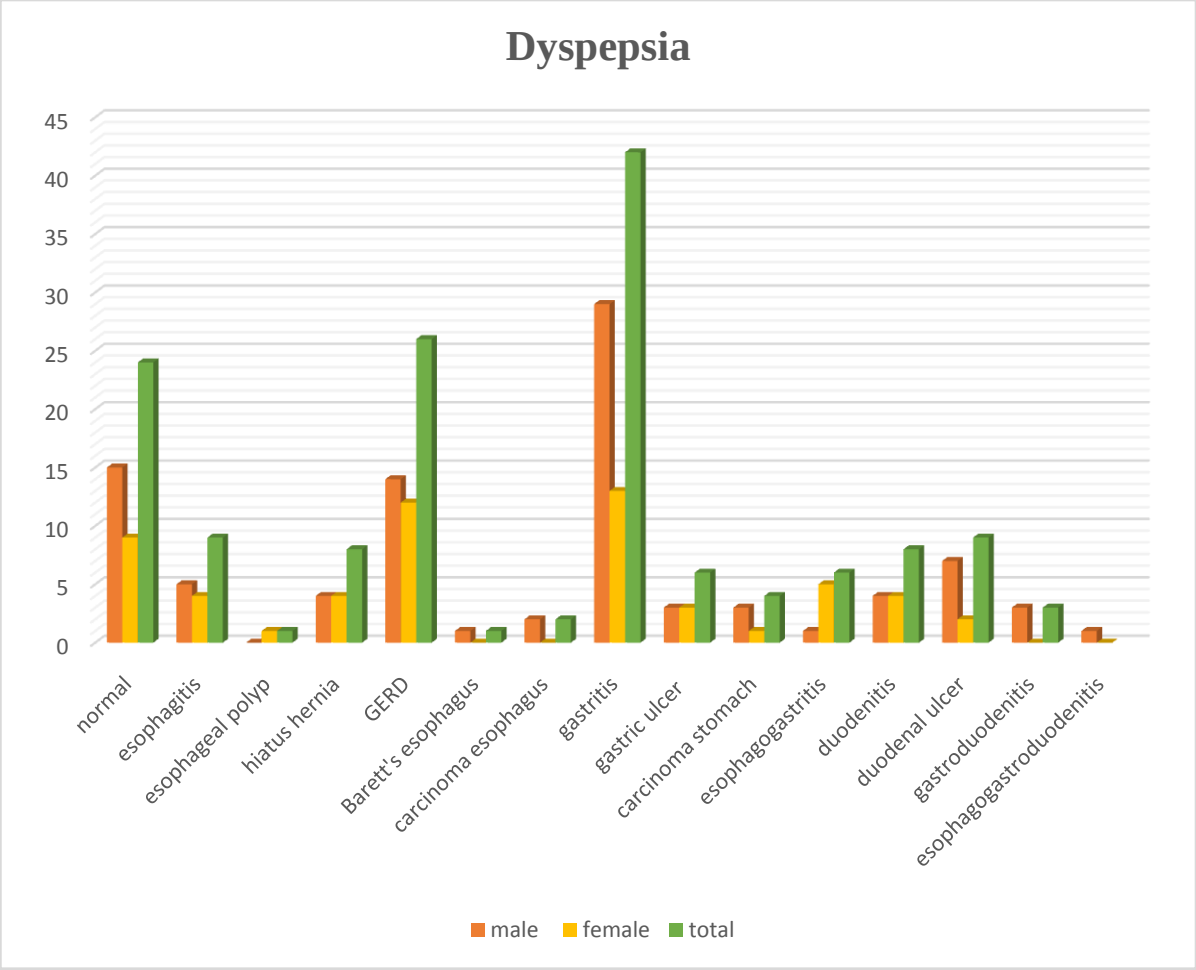
Data were collected and data analysis done.

RESULTS

Totally 150 patients were taken for the studyby convenience sampling method. Among which 92 were males and 58 were females. The patients were in age group ranging from 13 years to 86 years and the mean age was 43 years.

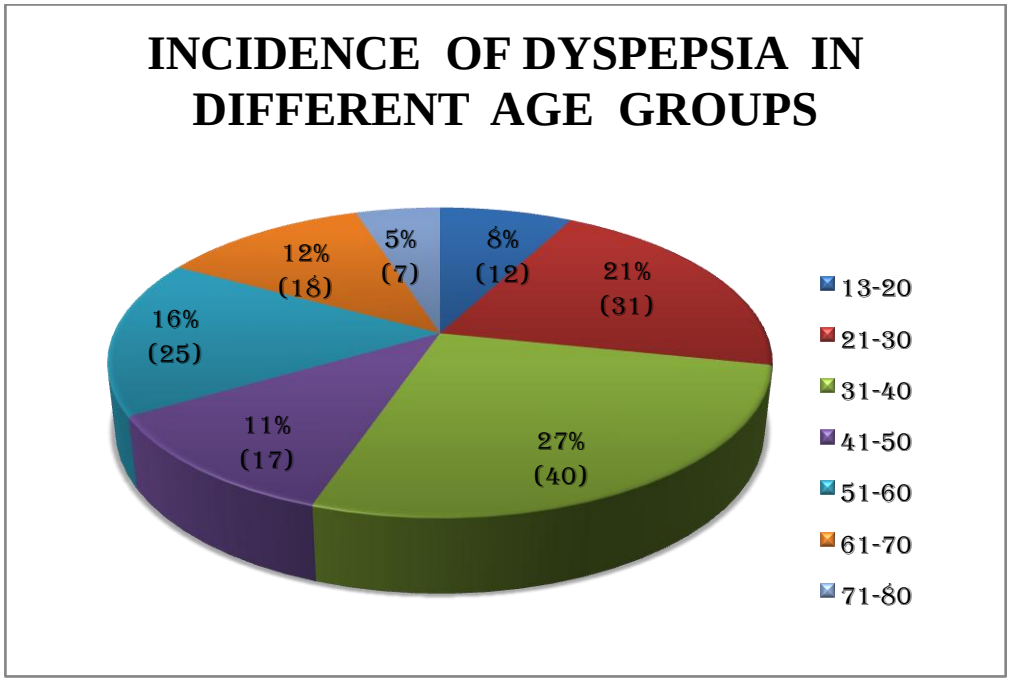
Dyspepsia

SI NO	Endoscopic findings	Male	Female	Total	Percentage
1	Normal	15	9	24	16%
2	Oesophagitis	5	4	9	6%
3	Oesophageal polyp	-	1	1	0.6%
4	Hiatus hernia	4	4	8	5.3%
5	GERD	14	12	26	17.3%
6	Barett's oesophagus	1	-	1	0.6%
7	Carcinoma oesophagus	2	-	2	1.3%
8	Gastritis	29	13	42	28%
9	Gastric ulcer	3	3	6	4%
10	Carcinoma stomach	3	1	4	2.6%
11	Oesophagitis	1	5	6	4%
12	Duodenitis	4	4	8	5.3%
13	Duodenal ulcer	7	2	9	6%
14	Gastroduodenitis	3	-	3	2%
15	oesophagogastroduodenitis	1	-	1	0.6%
	TOTAL	92	58	150	100%


Dyspepsia
45
40
35
30
25
20
15
10
5
0
normal
esophagitis
esophageal polyp
hiatus hernia
GERD
Barett's esophagus
carcinoma esophagus
gastritis
gastric ulcer
carcinoma stomach
esophagogastritis
duodenitis
duodenal ulcer
gastroduodenitis
esophagogastroduodenitis
male
female
total

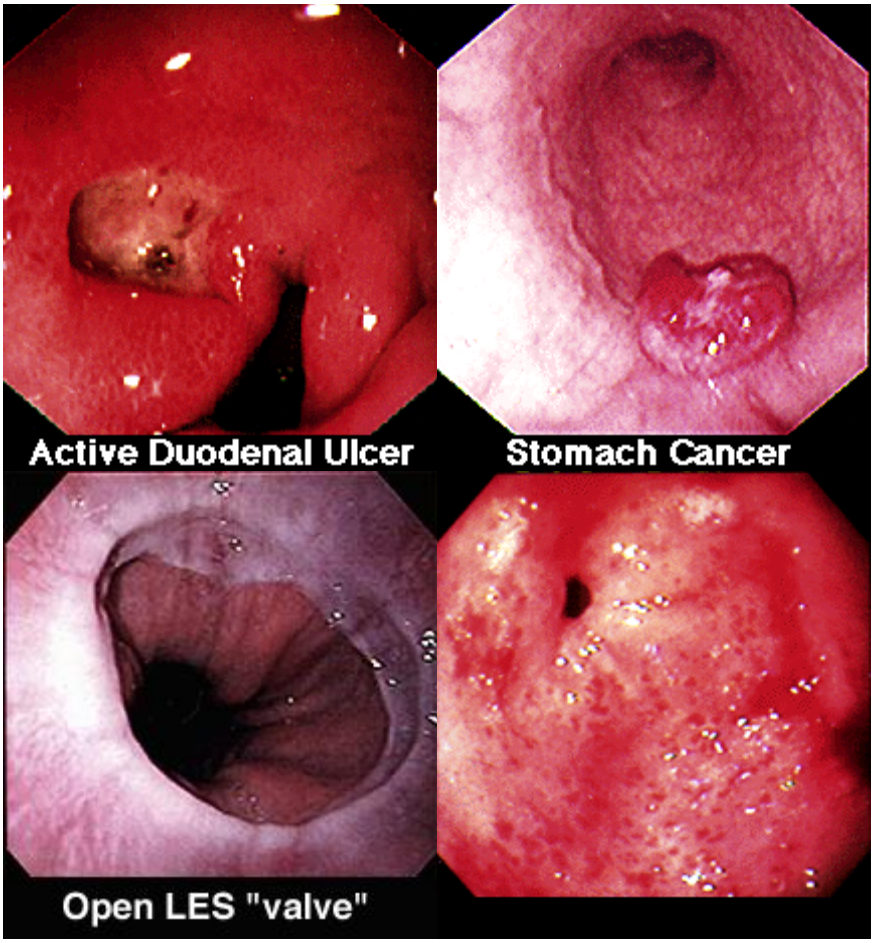
Incidence of dyspepsia in different age groups

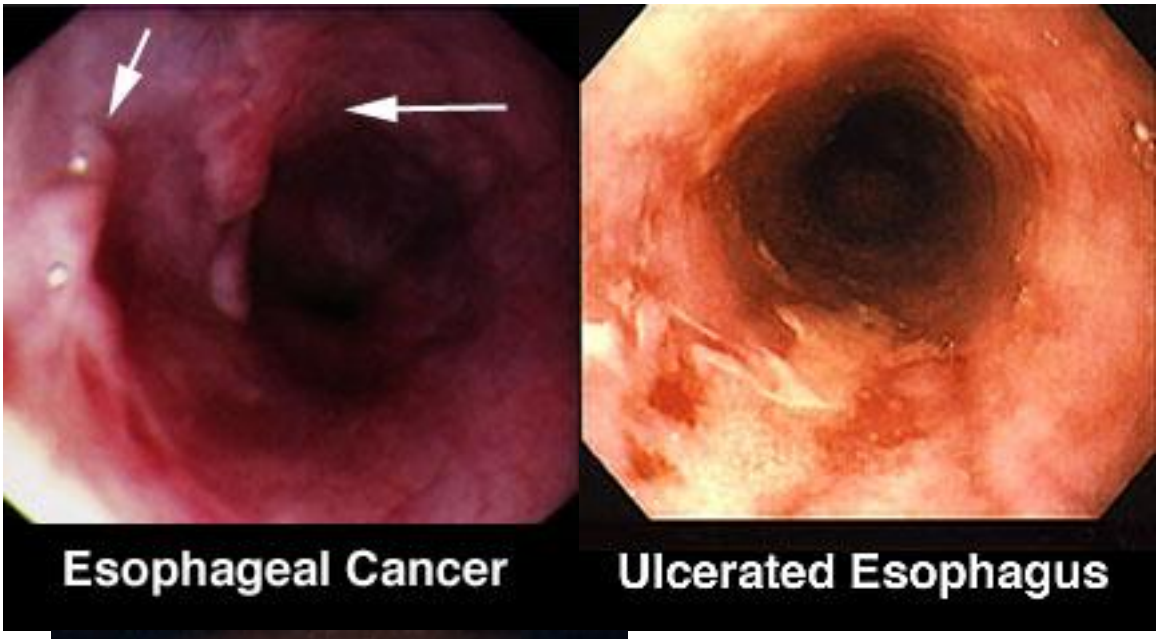
Age groups	No of cases
13-20	12
21-30	31
31-40	40
41-50	17
51-60	25
61-70	18
71-80	7

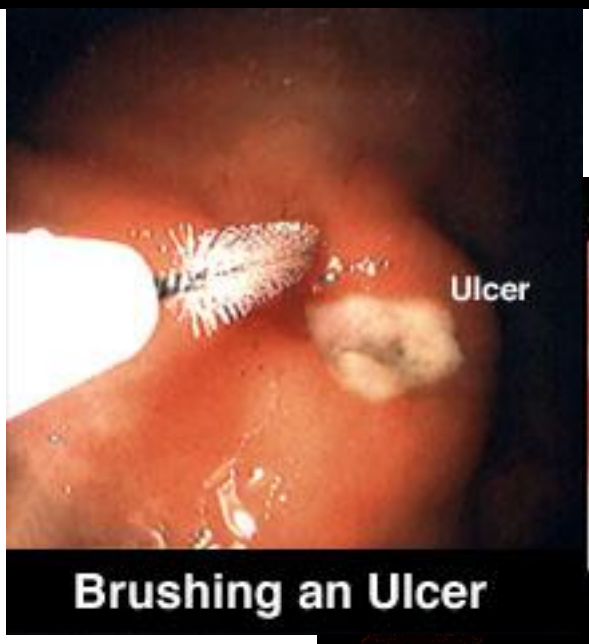


Upper gastro oesophago duodenoscopy was done for all patients.

Normal study was seen in 28 patients. This accounts for 18%. Gastritis was seen in 41 (29%) cases. This is the most common abnormal finding.GERD was seen in 26(18%) cases followed by duodenal ulcer and hiatus hernia in 8 cases (5.6%) each. Gastric ulcer and duodenitis in 7 cases(4.9%) each.




Esophageal Cancer
Ulcerated Esophagus


Ulcer
Brushing an Ulcer


Deep Stomach Ulcer

Carcinoma stomach was noted in 4 cases(2.8%) and duodenal ulcer was diagnosed in 2(1.4%) cases. Oesophageal polyp and Barrett's oesophagus in 1 case each.

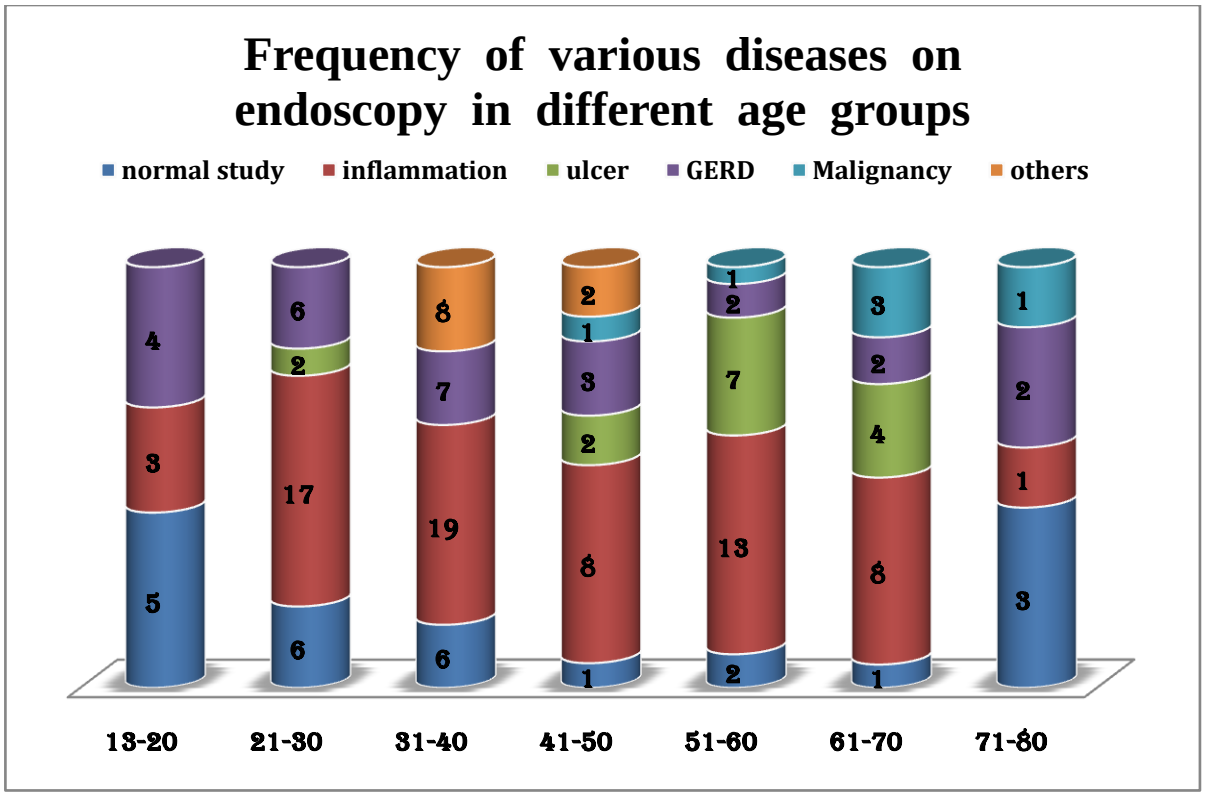
When the endoscopic findings were subdivided based on the age group significant findings were in seen in 21-40 years. GERD is seen mainly in age group of 31-40 years.

The inflammatory lesions were more common between 21-40 years of age.

Dyspepsia due to ulcer was common in age group of 41-70 years.

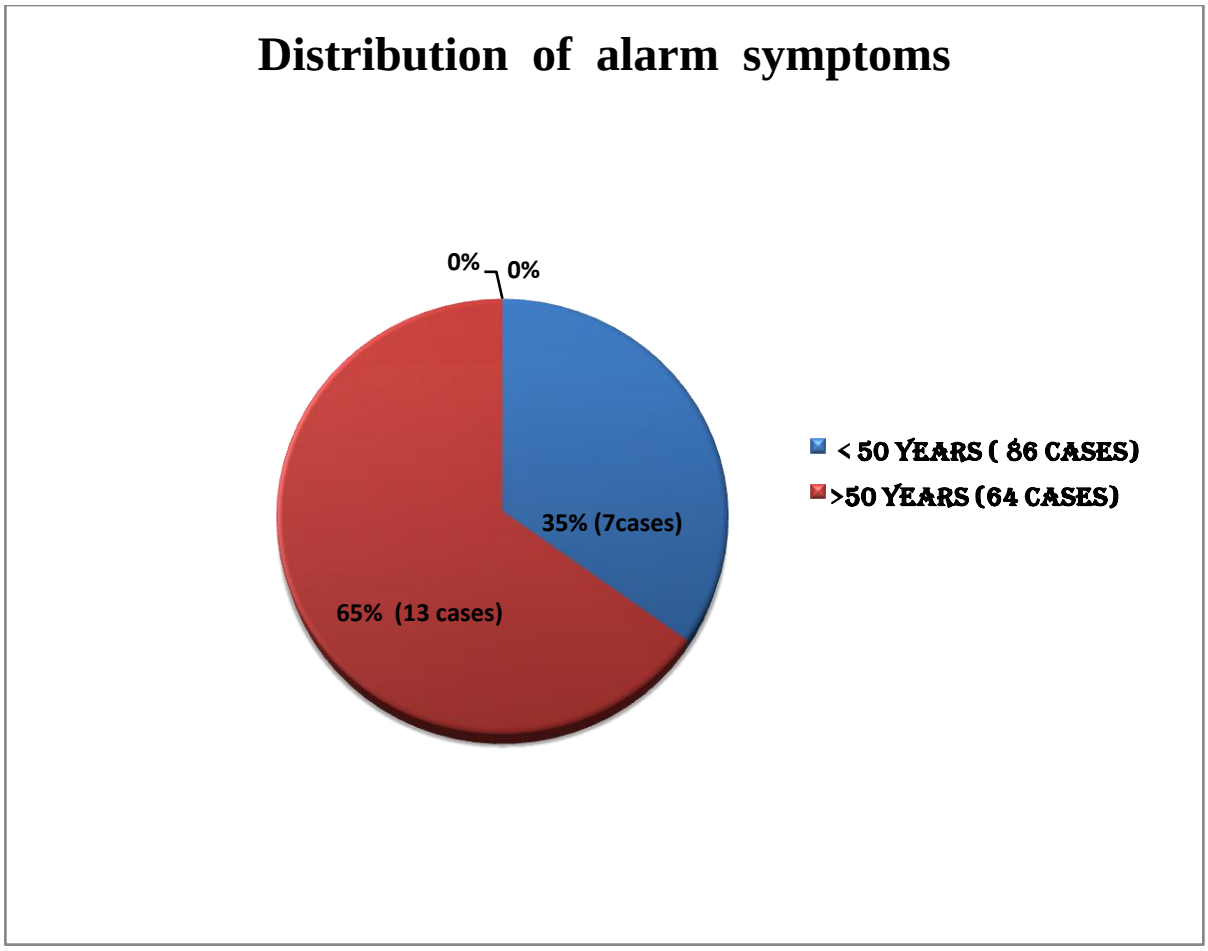
Frequency of various diseases on endoscopy in different age groups

Age groups	Normal study	inflammmation	ulcer	GERD	Malignancy	others	total	percentage
13-20	5	3	-	4	-	-	12	8%
21-30	6	17	2	6	-	-	31	20.6%
31-40	6	19	-	7	-	8	40	26.6%
41-50	1	8	2	3	1	2	17	11.3%
51-60	2	13	7	2	1	-	25	16.6%
61-70	1	8	4	2	3	-	18	12%
71-80	3	1	-	2	1	-	7	4.6%
total	24	69	15	26	6	10	150	100%



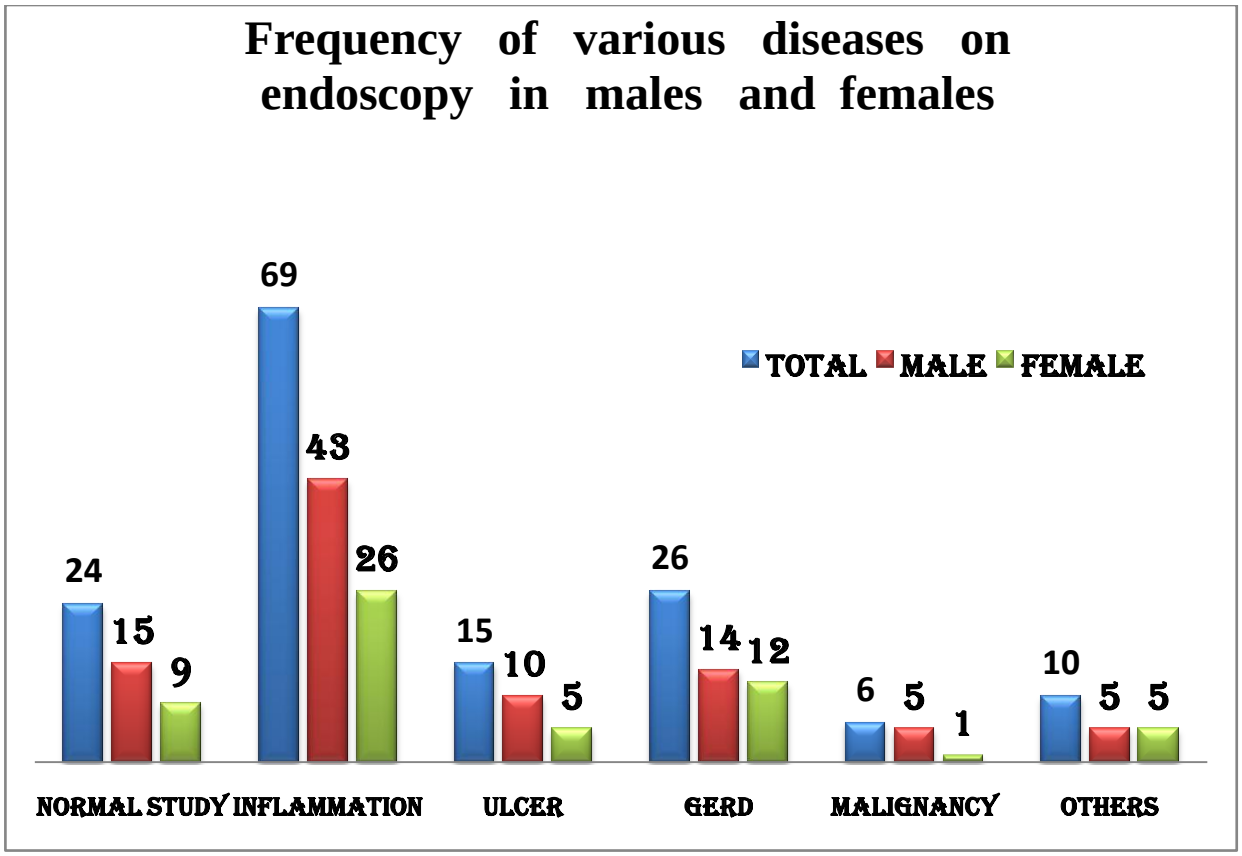
In this study both oesophageal and gastric malignancy were common above 50 years of age.

Among the 150 cases 20 cases had alarm symptoms.13 cases were above 50 years of age and 7 cases were below 50 years of age. Among the 6 malignant cases, 3 cases that is 50% had alarm symptoms



In case of dyspepsia, inflammatory lesion was the most common pathology.

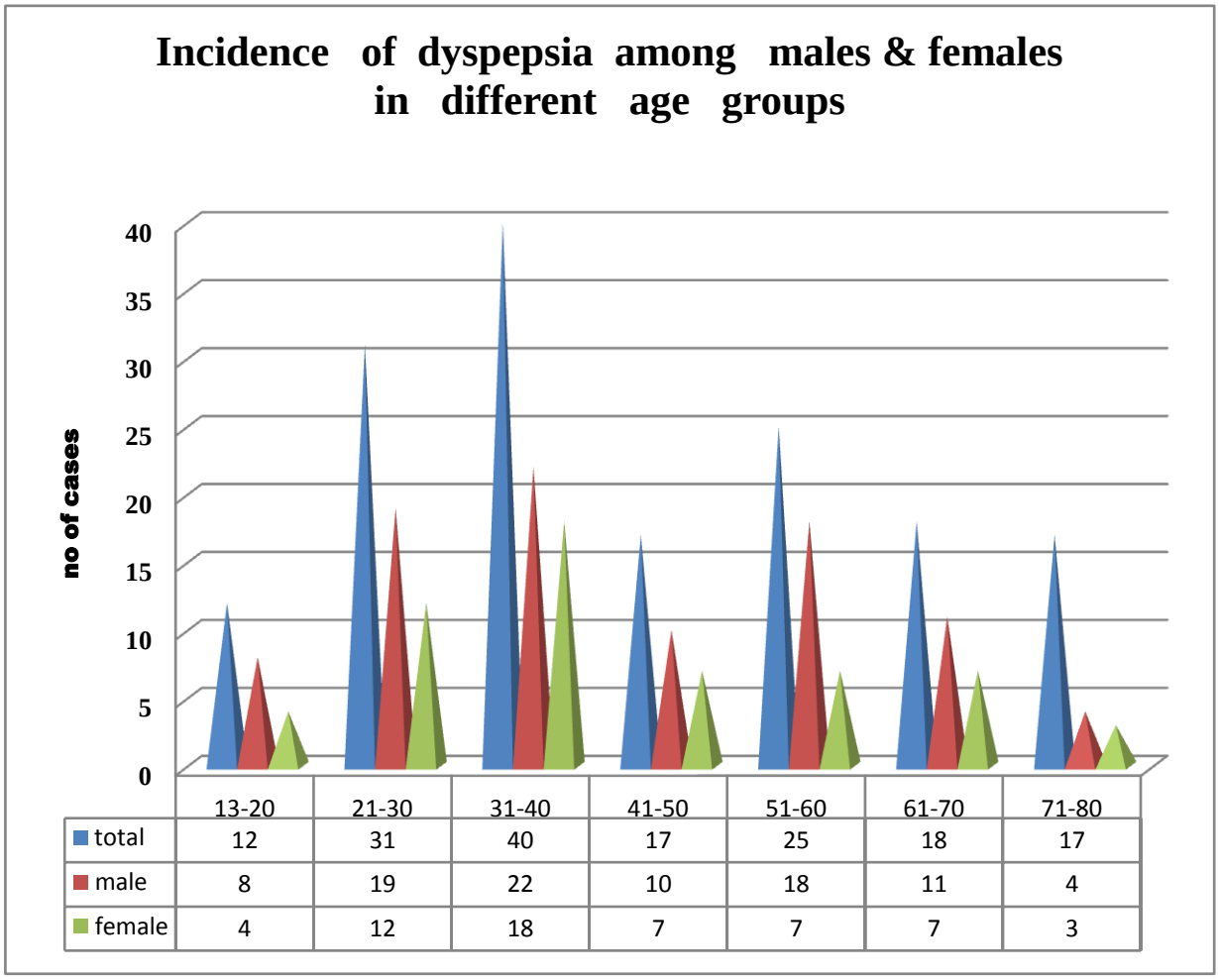
Around 68(47.1%), among which 42(48%) are males and females are 25(46%).



GERD was seen in 34 (24%) patients.18 cases among males and 16 among females

Dyspepsia due to ulcer was seen in 15 patients among which 10(10.4%) are males and 5(7.4%) are females.

Oesophageal and gastric malignancy were seen in 5(5.7%) cases, only in males.

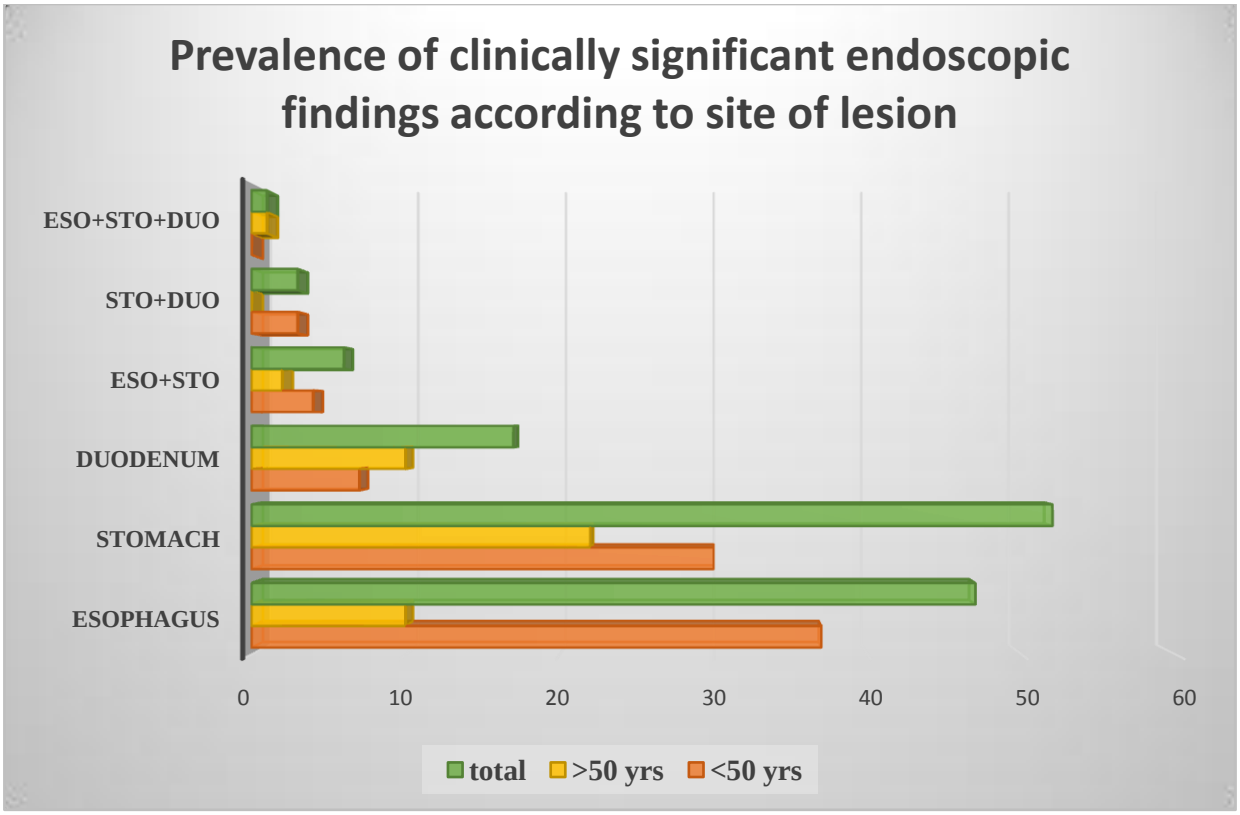


	13-20	21-30	31-40	41-50	51-60	61-70	71-80
total	12	31	40	17	25	18	17
male	8	19	22	10	18	11	4
female	4	12	18	7	7	7	3

Among the 122 cases with significant findings, 52(42%) cases had pathology in the stomach, 46 (38%)cases had pathology in the oesophagus and 15(12.5%) cases in the duodenum.

Prevalence of clinically significant endoscopic findings according to the site of lesions

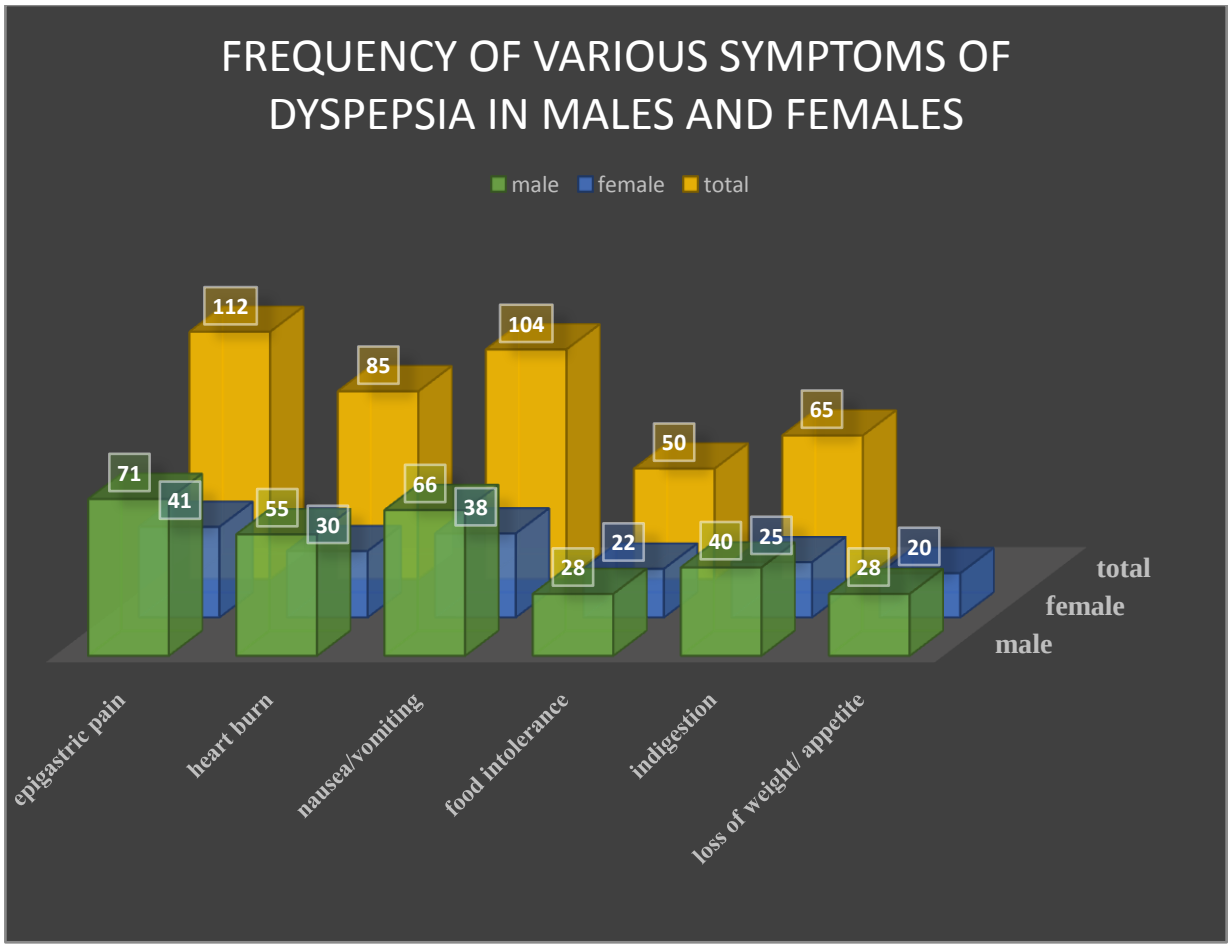
SI NO	CSF'S	Age<50 yrs	Age>50 yrs	Total	Percentage
1	Oesophagus	37	10	47	37.3%
2	Stomach	30	22	52	41.2%
3	Duodenum	7	10	17	13.4%
4	ESO+STO	4	2	6	4.7%
5	STO+DUO	3	-	3	2.3%
6	ESO+STO+DUO	-	1	1	0.7%
	Total	81	45	126	100%



Epigastric pain was the most common dyspeptic complaint seen in 112(78%) cases. Nausea and vomiting were seen in 104(72%) cases followed by heart burn in 85(60%) cases ,Indigestion in 65(45%) cases and loss of weight and appetite in 48(33.1%) cases

Frequency of various symptoms of dyspepsia in males and females

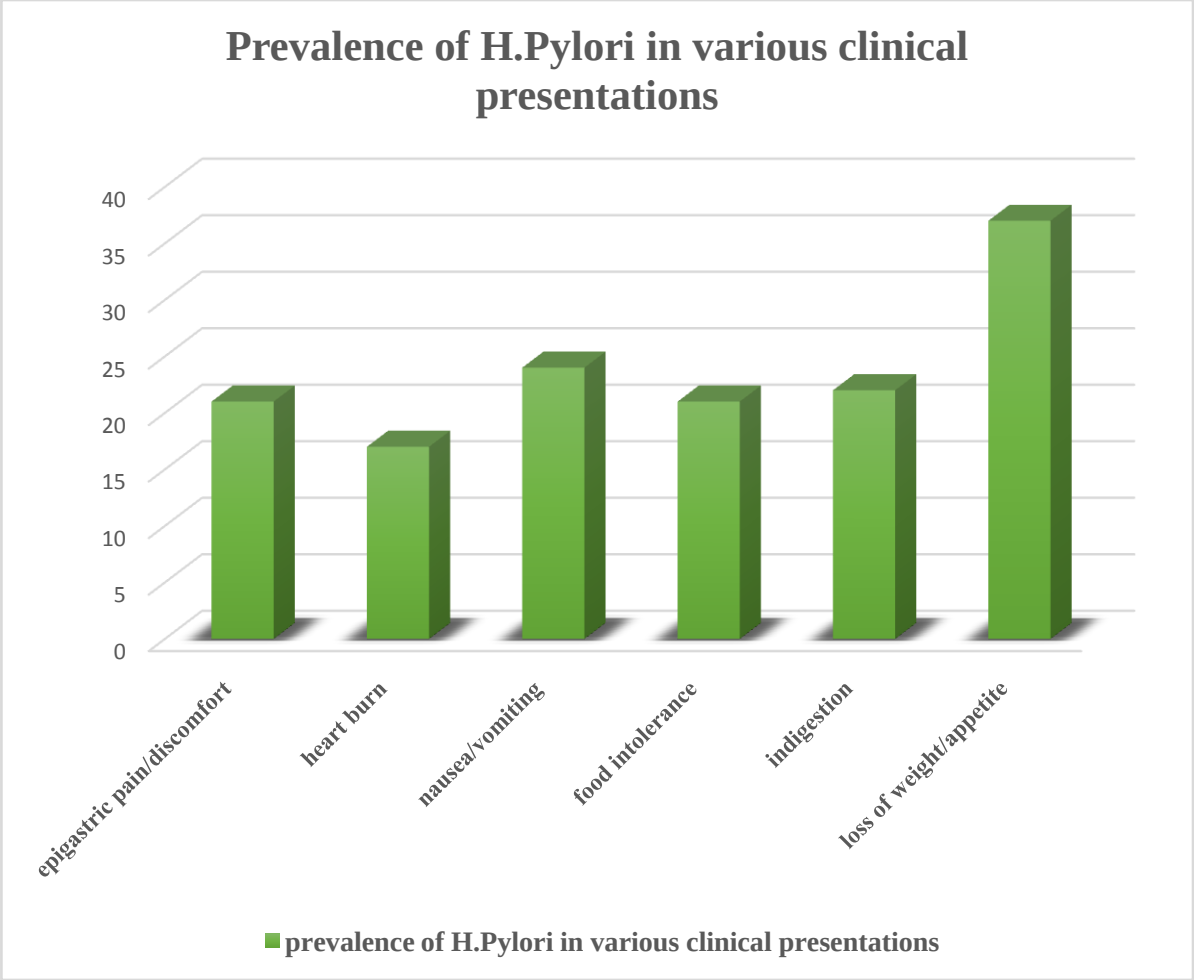
SI NO	Clinical presentation	male	female	total	percentage
1	Epigastric pain	71	41	112	74.6%
2	Heart burn	55	30	85	56.6%
3	Nausea/vomiting	66	38	104	69.3%
4	Food intolerance	28	22	50	33.3%
5	Indigestion	40	25	65	43.3%
6	Loss of weight/appetite	28	20	48	32%



The common clinical finding in H pylori positive cases was loss of weight and appetite(63%) followed by nausea and vomiting(42%). Epigastric discomfort was seen only in 36.6% cases.

Prevalence of H.Pylori in various in various clinical presentations

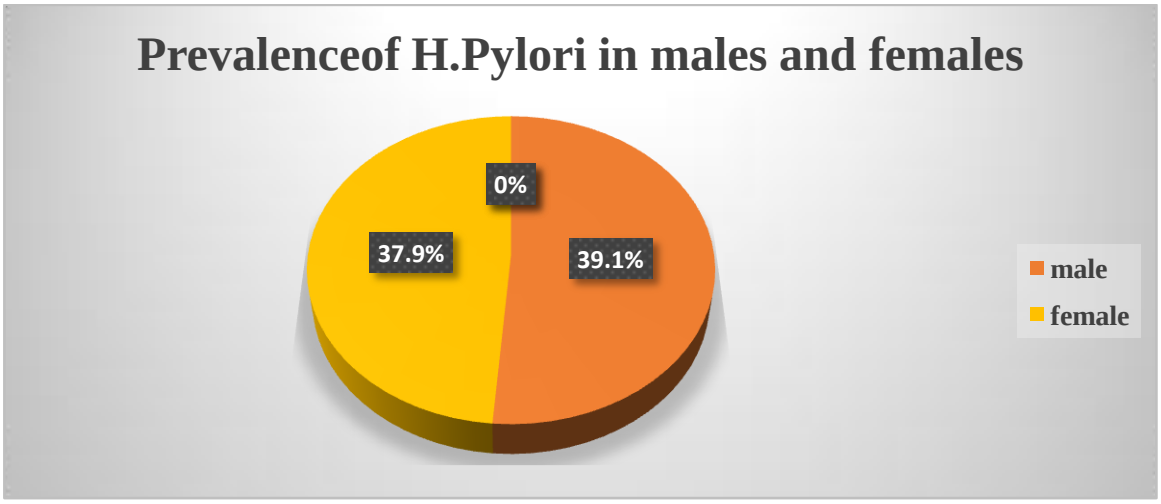
Clinical presentation	No of cases	H.Pylori positive	percentage
Epigastric pain/discomfort	112	21	36.2%
Heart burn	85	17	29.4%
Nausea/vomiting	104	24	41.3%
Food intolerance	50	21	37.1%
Indigestion	65	22	37.9%
Loss of weight/appetite	48	21	36.2%



H pylori prevalence is slightly more in male patients(39.1%) when compared to females(37.9%).

Prevalence of H.Pylori in males and females

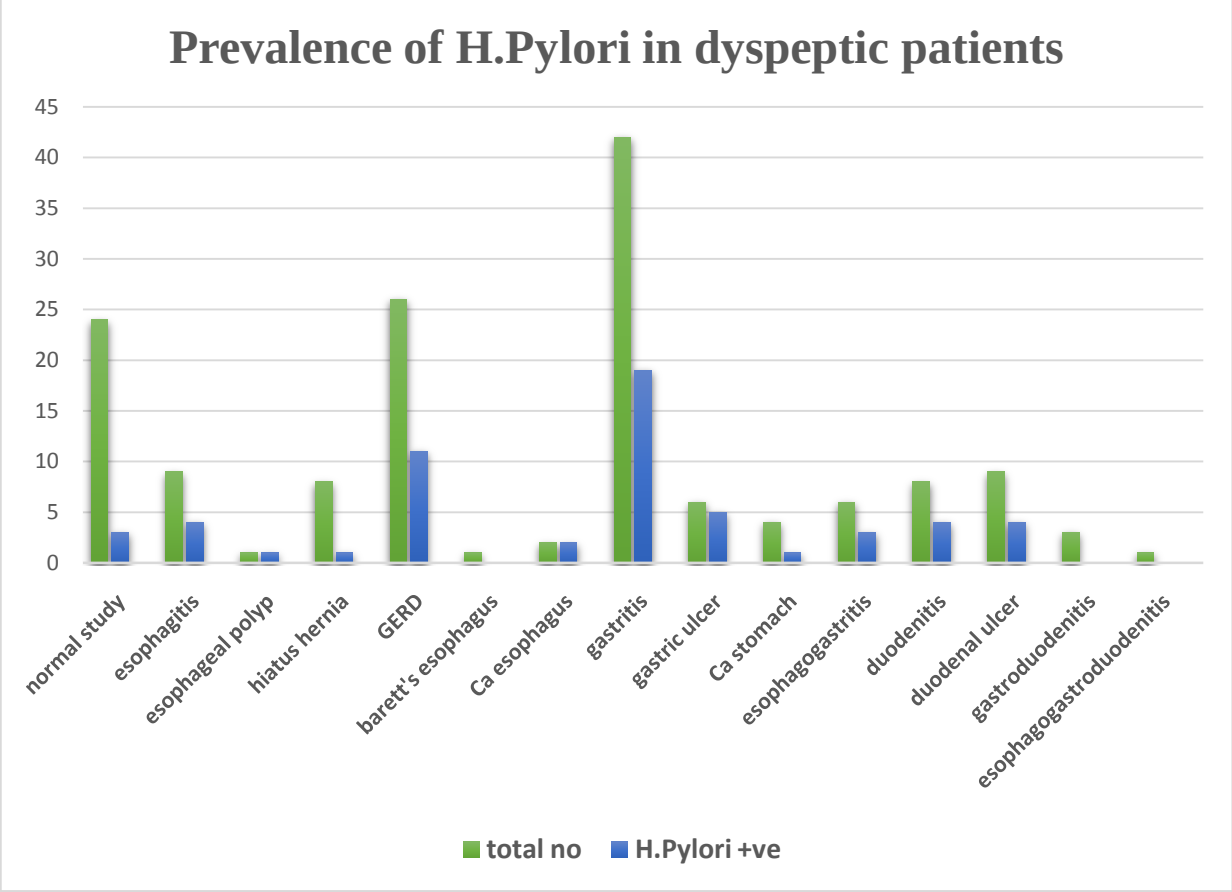
Gender	Total	H.Pylori positive	Percentage
Male	92	36	39.1%
Female	58	22	37.9%
total	150	58	38.6%



Among the 28 patients with normal endoscopic study, 5 patients were positive for H pylori on rapid urease test.

Although only 39% of dyspeptic patientswere associated with H pylori, it was 100% positive in patients with oesophagogastroduodenitis followed by 75% in case of duodenal ulcer.

In gastric and oesophageal carcinoma, 50% of patients were positive in this study.



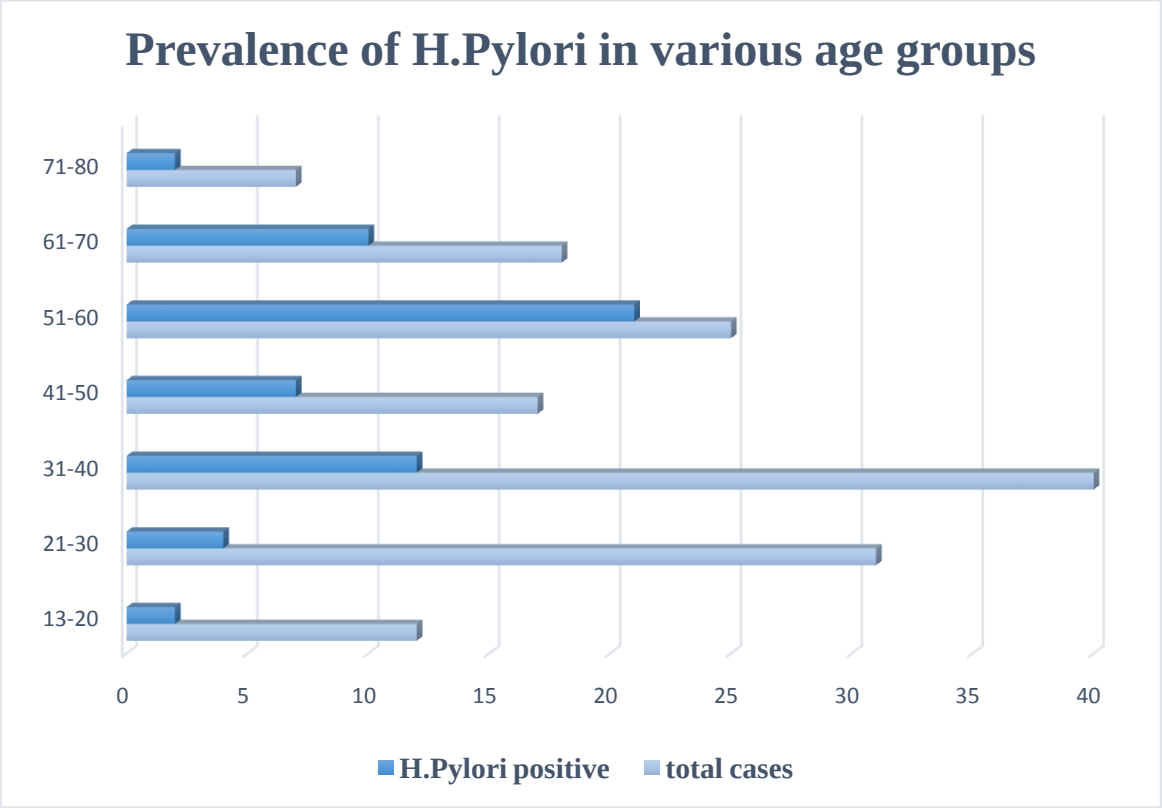
Prevalence of H.Pylori in dyspeptic patients

SI NO	Endoscopic findings	Total no	H.Pylori positive	Percentage
1	Normal study	28	5	12.5%
2	Oesophagitis	9	4	44.4%
3	Oesophageal polyp	1	1	100%
4	Hiatus hernia	8	1	12.5%
5	GERD	26	11	42.3%
6	Barett‘s oesophagus	1	-	-
7	Carcinoma oesophagus	2	1	50%
8	Gastritis	42	19	45.2%
9	Gastric ulcer	6	5	83.3%
10	Carcinoma stomach	4	2	50%
11	Oesophagogastritis	6	3	50%
12	Duodenitis	8	4	50%
13	Duodenal ulcer	9	4	44.4%
14	Gastroduodenitis	3	-	-
15	Oesophagogastroduodenitis	1	-	-
	Total	150	58	38.7%

H pylori was more in the 51-60 years age group and least common in the 13-20 years age group.

Prevalence of H.Pylori in various age groups

SI NO	Age groups	Total no	H.Pylori positive	Percentage
1	13-20	12	2	16.6%
2	21-30	31	4	12.9%
3	31-40	40	12	30%
4	41-50	17	7	41.17%
5	51-60	25	21	84%
6	61-70	18	10	55.5%
7	71-80	7	2	28.5%
	total	150	58	38.6%


Prevalence of H.Pylori in various age groups
71-80
61-70
51-60
41-50
31-40
21-30
13-20
0
5
10
15
20
25
30
35
40
H.Pylori positive
total cases

DISCUSSION

A clinical prospective study on ‘Role of endoscopy in patients presenting with dyspepsia’ at Kilpauk Medical College, Chennai to study the usefulness of endoscopy in patients presenting with dyspepsia.

Dyspepsia is a broad spectrum of symptoms with many differential diagnosis. So many persons have dyspeptic symptoms and it is practically difficult to perform endoscopy in all patients with dyspepsia taking into account the cost and man power.

Hence this thesis is to study the appropriate role of endoscopy in management of dyspepsia.

Observations made in the study was comparable to many other studies in the past. Previous studies has indicated that endoscopy is not needed for simple dyspepsia in younger age group as initial diagnostic tool.

The study was done in 150 cases after getting their written consent for the procedure and the study.The study period was 10 months from October 2013 to July 2014. All the patients included in the study were subjected to upper oesophago gastroduodenoscopy and their findings were analysed.

CLINICAL PRESENTATION

Most of the patients had epigastric pain or discomfort as their main complaint. It was present in nearly 114(79%) patients. Epigastric pain was followed by nausea and vomiting presenting in 104(73%) patients. Heart burn was the presenting complaint in 85 patients and indigestion in 68 patients.

In the study conducted by Thomson ABR et al epigastric pain was present in 34.3% of the study population followed by heart burn in 24.5%. This was quite comparable to our study regarding presenting complaints.

COMPARISON OF AGE DISTRIBUTION

Most of the patients in the study were in the age of 31-40 years. Mean age in the study population was 43 years.

The mean age in previous similar studies is shown the table

Thus the mean age when compared with other studies was almost similar.

Comparison of age distribution

SI NO	Name of study	Mean age in years
1	**Thomson A B R et al**	**45.6**
2	**Ziauddin**	**42.2+/-15.7**
3	**Choomsri P et al**	**41**
4	**Present study**	**43**

COMPARISON OF GENDER DISTRIBUTION

Nearly 62% of study population were male and 38% were female. The various presentations of dyspepsia were common in males than females expect oesophago gastritis which were more common in females.

Other similar studies that various presentation were common in males than females. In study conducted by Khan N et al male to female ratio was 2.3:1 and in study conducted by Ziauddin ratio was 1.6 : 1.

But in one of the Australian based studies, females with dyspepsia were more than males.

COMPARISON OF VARIOUS ENDOSCOPIC FINDINGS

SI NO	Name of the study	Gastritis	Reflux esophagitis/ GERD
1	Sarwar et al	13%	20%
2	Ziauddin	18%	14%
3	Present study	28.9%	18.3%

Nearly 122 patients among 150 patients in our study presented with significant endoscopic findings. Inflammatory lesion was the most common endoscopic Finding, among which gastritis was the most common, accounting for 28.9% ,followed by GERD. In 18.3% oesophagitis, hiatus hernia and duodenal ulcer accounted for 8% each.

The incidence of gastritis was more when compared to other studies like studies by Sarwar et al and Ziauddin et al. But the incidence of GERD was comparable with study conducted by Sarwar et al.

PREVALENCE OF HELICOBACTER PYLORI

The H pylori incidence was more common in 51-60 years age group and least common in 13-20 years. This shows that the incidence of H pylori increases with age. The incidence of H pylori in males and females were equal and showed no significant difference.

On comparing the clinical presentation with H pylori, surprisingly loss of weight and appetite was present in nearly 63% of cases. The next common symptom was nausea and vomiting present in 41.3% followed by indigestion(38.5%) and epigastric discomfort(36.6%).

The incidence of H pylori in our study was 38.7%.in study conducted by Thomson et al it was 30% and Choomsri P et al was 23%

Prevalence of H.Pylori in dyspeptic patients

SI NO	STUDIES	H.Pylori positive in percentage
1	Choomsri P et al	23%
2	Thomson ABR et al	30%
3	Present study	38.7%

The incidence was highwhen compared to other studies. As people under study were from lower economic status this could be the reason for high incidence of H pylori in our study.

When H pylori prevalence was compared with various endoscopic findings, H pylori was seen in 53.6% (22/41) of patients with gastritis and 71% (6/8) with duodenal ulcer and 60% (3/5) with gastric ulcer[50.]

Prevalence of H.Pylori in various studies and their endoscopic findings

SI NO	Name of study	gastritis	GU	DU	CA stomach
1	Mustapha SK et al	89.1%	61.9%	100%	33.3%
2	Marshall et al	54.2%	81.8%	100%	-
3	Wulfen et al	62.2%	72.2%	83.3%	-
4	Vaira et al	57.6%	83%	92%	87%
5	Present study	53.6%	60%	71%	50%

H ELICOBACTER PYLORI IN SMOKERS

Among the 92 males, 53 were smokers and 43 of them, i.e 82% were positive for H pylori and among 39 non smokers ,23 , i.e 55% were positive for H pylori.

HELICOBACTER PYLORI AND LYMPHOID FOLLICLES

Lymphoid follicles are commonly present in H pylori positive cases. In previous studies it ranges between 27% to 100%. So it was concluded previously that lymphoid follicles is a strong predictor of H pylori infection.

ALARM SYMPTOMS AND ENDOSCOPIC FINDINGS

Alarm symptoms were present in 20 patients and 17 among them had significant endoscopic findings. The most important correlation was that among the 4 patients with gastric carcinoma 3 of them had alarm symptoms.

COMPARISON OF INCIDENCE OF GASTRIC CARCINOMA

Four patients among the 150 patients were found to have gastric malignancy. Among the four, three were male patients and were above 50 years of age. The incidence of gastric carcinoma in study conducted by Khan et al was 3%, Choomsri et al was 1% and Ziauddin et al was 4%.Thus present study was comparable to other studies.

Comparison of incidence of gastric malignancies

SI NO	NAME OF STUDY	Percentage of gastric malignancies
1	Choomsri P et al	1%
2	Khan N et al	3%
3	Ziauddin	4%
4	Present study	2.67%

SUMMARY

The study on ‘The role of endoscopy in patients presenting with dyspepsia’ is a prospective clinical study conducted on 150 patients with dyspeptic symptoms in Kilpauk Medical College, Chennai.

Patients included in the study were first interviewed for various dyspeptic symptoms including alarm symptoms. Then all the patients were subjected to endoscopy and based on the endoscopic findings patients were subjected to various tests - endoscopic biopsy, histopathological examination, rapid urease test. The results were analysed.

Significant endoscopic findings were seen in 84.5% of the study group.Gastritis and GERD was present in majority of the cases. Patients with simple dyspepsia did not have serious lesions in majority of the cases during endoscopy. Mucosal inflammatory lesions were seen in patients positive for H pylori organism. Gastric or oesophageal malignancy was present mostly in patients above 50 years of age and 50% of them had alarm symptoms. Only 1 person among the 150 cases, below 50 years of age was diagnosed to have gastric carcinoma

Hence the study shows that endoscopy is not required in patients less than 50 years of age with no alarm symptoms.Testing for H pylori for all dyspeptic patientsand

starting on anti H pylori regimen was cost effective compared to the initial endoscopy for dyspeptic patients.

CONCLUSION

Based on the present study on "The role of endoscopy in dyspepsia symptom complex" done in Kilpauk medical college, we conclude that-

- Prevalence of dyspepsia was more in 31-40 years age group and more common in males than females.
- Epigastric pain or discomfort is the most common presenting symptom in dyspeptic patients
- Gastritis was the most common endoscopic finding
- Among the study population H pylori prevalence was 39%
- Incidence of malignancy among the study population was 4%
- Among the 6 malignant positive cases 5 were above 50 years of age
- Endoscopy should be recommended for patients presenting with dyspepsia with alarm symptoms as the correlation between alarm symptoms of dyspepsia and gastric lesions were significant
- In patients without alarm symptoms below 50 years of age there was no significant endoscopic findings. So these patients should be treated initially rather than subject them to endoscopy.
- H pylori prevalence was more in age group above 50 years than younger age group thus H pylori infection increases with age

- Smoking is an important risk factor for H pylori infection.

- Presence of lymphoid follicles in histopathology significantly correlates with H pylori infection

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MASTER CHART

S NO	NAME	AGE	GENDER	OP NO	CLINICAL FEATURES						SIGNS		ENDOSCOPIC FINDINGS	H.PYLORI	HPe
					EP/D	N/V	HB	FI	IDG	LW/A	PA	ET			
1	**Rajesh**	**26**	**M**	**1214**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GDs**	**-**	**ACG**
2	**Palanisamy**	**61**	**M**	**3243**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
3	**Saritha**	**19**	**F**	**2267**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**NS**	**-**	**NSP**
4	**Giridharan**	**52**	**M**	**3344**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**Ds**	**+**	**HPG**
5	**Thiagu**	**44**	**M**	**1678**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**BE**	**-**	**ACG**
6	**Ramesh**	**21**	**M**	**2456**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**NS**	**-**	**NSP**
7	**Leelavathy**	**58**	**F**	**1890**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**DU**	**+**	**CPDs**
8	**Irudhayaraj**	**32**	**M**	**2432**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**HH**	**-**	**NSP**
9	**Krishnan**	**34**	**M**	**2865**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**Es**	**-**	**EE**
10	**Lakshmi**	**72**	**F**	**2236**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**GERD**	**+**	**RE**
11	**Shankar**	**28**	**M**	**3658**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**DU**	**-**	**CPGs**
12	**Radha**	**31**	**F**	**2876**	**Y**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Es**	**-**	**RE**
13	**Mariappan**	**54**	**M**	**1457**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**Es**	**+**	**EE**
14	**Janaki**	**33**	**F**	**1008**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**EGs**	**+**	**CGs**
15	**Ravi**	**18**	**M**	**1577**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Ds**	**-**	**CPDs**
16	**Vandhana**	**31**	**F**	**2190**	**N**	**N**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**Gs**	**+**	**HPG**
17	**Venkatesh**	**34**	**M**	**3454**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**GDs**	**-**	**CGs**
18	**Ramesh**	**28**	**M**	**2545**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**DU**	**-**	**CPDs**
19	**Kamatchi**	**59**	**F**	**1967**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**GU**	**+**	**ACG**

20	**Bhavani**	**69**	**F**	**2756**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**EGs**	**+**	**HPG**
21	**Vijay**	**17**	**M**	**1354**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**NS**	**-**	**NSP**
22	**Latha**	**33**	**F**	**1788**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
23	**Kowsar**	**60**	**M**	**2436**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Es**	**+**	**RE**
24	**Daniel**	**28**	**M**	**3867**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
25	**Vijaya**	**35**	**F**	**1089**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**Gs**	**-**	**CGs**
26	**Prakasham**	**32**	**M**	**1577**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
27	**Shobana**	**19**	**F**	**3787**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
28	**Dhanapal**	**51**	**M**	**3554**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
29	**Asokan**	**50**	**M**	**1879**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**DU**	**+**	**CPDs**
30	**Kannan**	**25**	**M**	**3578**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**GERD**	**-**	**RE**
31	**Varadharaj**	**63**	**M**	**2454**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**GERD**	**-**	**RE**
32	**Ganeshan**	**42**	**M**	**1788**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GU**	**-**	**ACG**
33	**Sudhandira**	**32**	**F**	**3566**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**HH**	**+**	**HPG**
34	**Geetha**	**34**	**F**	**1214**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
35	**Priyanka**	**27**	**F**	**1345**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**-**	**CGs**
36	**Anandan**	**30**	**M**	**1675**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**GERD**	**+**	**RE**
37	**Sugumari**	**33**	**F**	**2547**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**Gs**	**+**	**HPG**
38	**Kanagu**	**68**	**M**	**2798**	**Y**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**N**	**DU**	**-**	**CPD**
39	**Subashini**	**25**	**F**	**1808**	**Y**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Gs**	**-**	**CGs**
40	**Shanthi**	**41**	**F**	**2586**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
41	**Saraswathi**	**49**	**F**	**1898**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
42	**Neelakandan**	**53**	**M**	**3564**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Gs**	**+**	**HPG**
43	**Gopalan**	**78**	**M**	**1303**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**EGDs**	**-**	**EE**

44	**Karthik**	**16**	**M**	**2342**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**NS**	**-**	**NSP**
45	**Ramani**	**35**	**F**	**1896**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
46	**Hajeera**	**29**	**F**	**1635**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**GERD**	**-**	**RE**
47	**Kaliammal**	**67**	**F**	**1457**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Es**	**+**	**EE**
48	**Ramkumar**	**46**	**M**	**2277**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**GERD**	**+**	**RE**
49	**Rakesh**	**15**	**M**	**2785**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**EGs**	**-**	**CGs**
50	**Suganya**	**24**	**F**	**3688**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**GERD**	**+**	**NSP**
51	**Krishnaveni**	**62**	**F**	**1894**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**DU**	**-**	**CPD**
52	**Moorthy**	**55**	**M**	**3746**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GU**	**+**	**ACG**
53	**Subathra**	**43**	**F**	**2786**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
54	**Ashok**	**29**	**M**	**2576**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
55	**Manimoli**	**21**	**F**	**3454**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**GERD**	**-**	**NSP**
56	**Manikandan**	**58**	**M**	**2344**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
57	**Meenakshi**	**68**	**F**	**2143**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**Y**	**N**	**CaS**	**-**	**Sig r c**
58	**Kishore**	**20**	**M**	**1565**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**NSP**
59	**Rani**	**64**	**F**	**2435**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**Y**	**GU**	**+**	**ACG**
60	**Sundaram**	**56**	**M**	**3534**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
61	**Malarvizhi**	**45**	**F**	**3241**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**EP**	**+**	**Hyp P**
62	**Dinesh**	**25**	**M**	**2236**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**CGs**
63	**Manishkumar**	**36**	**M**	**1756**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**HH**	**-**	**NSP**
64	**Natarajan**	**40**	**M**	**1543**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Gs**	**+**	**HPG**
65	**Divya**	**24**	**F**	**1673**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
66	**Ravikumar**	**52**	**M**	**3435**	**N**	**N**	**Y**	**N**	**N**	**Y**	**Y**	**N**	**GU**	**+**	**HPG**
67	**George**	**77**	**M**	**2565**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**+**	**RE**

68	**Hariharan**	**42**	**M**	**2563**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
69	**Govindan**	**44**	**M**	**3285**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Es**	**+**	**EE**
70	**Yasaar**	**30**	**M**	**1134**	**N**	**N**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**Gs**	**-**	**ACG**
71	**Akila**	**16**	**F**	**1644**	**Y**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**N**	**NS**	**-**	**NSP**
72	**Jayanthi**	**31**	**F**	**2853**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
73	**Sekar**	**62**	**M**	**1674**	**N**	**N**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**CaE**	**+**	**Sq c c**
74	**Devanderan**	**33**	**M**	**2753**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**N**	**NS**	**-**	**CGs**
75	**Karthikeyan**	**24**	**M**	**1134**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
76	**Anbalagi**	**47**	**F**	**1672**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
77	**Babu**	**60**	**M**	**1965**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**NS**	**+**	**CGs**
78	**Nazeer**	**25**	**M**	**2787**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**NS**	**-**	**NSP**
79	**Saravanan**	**65**	**M**	**2674**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**Y**	**N**	**CaS**	**-**	**Ade c**
80	**Nandhini**	**32**	**F**	**2875**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**NS**	**+**	**CGs**
81	**Jayashree**	**29**	**F**	**2974**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
82	**Thangavel**	**59**	**M**	**1674**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
83	**Guna**	**35**	**M**	**3236**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**NSP**
84	**Seetha**	**72**	**F**	**2564**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**NS**	**-**	**CGs**
85	**Venkatesh**	**27**	**M**	**3854**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**N**	**Gs**	**+**	**HPG**
86	**Sudhakar**	**43**	**M**	**2632**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
87	**Subbaih**	**63**	**M**	**2743**	**N**	**N**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**Gs**	**+**	**HPG**
88	**Gnanasekar**	**37**	**M**	**1965**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
89	**Premkumar**	**18**	**M**	**2113**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**GERD**	**+**	**RE**
90	**Vaniammal**	**60**	**F**	**1920**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Ds**	**+**	**HPG**
91	**Valli**	**70**	**F**	**1056**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**NSP**

92	**Vijayakumar**	**34**	**M**	**3452**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
93	**Manokar**	**56**	**M**	**2433**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**GERD**	**+**	**RE**
94	**Minnoli**	**24**	**M**	**3754**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
95	**Arulmozhi**	**53**	**F**	**2455**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**NS**	**+**	**CGs**
96	**Vijaya**	**57**	**F**	**1674**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**EGs**	**-**	**CGs**
97	**Pawlina**	**64**	**M**	**3545**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**Gs**	**+**	**ACG**
98	**Sarasu**	**74**	**M**	**1212**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
99	**Mythili**	**24**	**F**	**2132**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**EGs**	**-**	**NSP**
100	**Victoria**	**41**	**F**	**1865**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**N**	**EGs**	**+**	**HPG**
101	**Bharathi**	**36**	**F**	**2667**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**HH**	**-**	**CGs**
102	**Maheshwari**	**38**	**F**	**2864**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Gs**	**+**	**HPG**
103	**Bhoopathy**	**36**	**M**	**1865**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
104	**Vignesh**	**23**	**M**	**2896**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**CGs**
105	**Thiruvaram**	**58**	**F**	**1087**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**	**GERD**	**+**	**RE**
106	**Jayagar**	**22**	**M**	**1554**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
107	**Sanamal**	**56**	**F**	**2976**	**N**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**Gs**	**+**	**HPG**
108	**Vishnu**	**22**	**M**	**1564**	**Y**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**N**	**Gs**	**-**	**CGs**
109	**Prabakar**	**45**	**M**	**2075**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**CaE**	**+**	**Ade c**
110	**Dilipan**	**37**	**M**	**2176**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**Gs**	**-**	**ACG**
111	**Rukumani**	**74**	**F**	**2396**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
112	**Sulaiman**	**35**	**M**	**2455**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
113	**Balaji**	**18**	**M**	**1574**	**Y**	**N**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**NSP**
114	**Chelappa**	**61**	**M**	**3765**	**N**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**Ds**	**+**	**CPDs**
115	**Kirubakaran**	**55**	**M**	**2753**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**DU**	**+**	**CPDs**

116	**Rajadurai**	**39**	**M**	**1325**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**HH**	**-**	**CGs**
117	**Peter**	**24**	**M**	**1465**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Es**	**-**	**EE**
118	**Thirupathi**	**59**	**M**	**1921**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
119	**Vaishnavi**	**23**	**F**	**2121**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Es**	**-**	**RE**
120	**Sureshkumar**	**46**	**M**	**1422**	**Y**	**N**	**N**	**N**	**Y**	**N**	**N**	**N**	**Ds**	**-**	**CPD**
121	**Jayabalan**	**66**	**M**	**1889**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
122	**Sakthivel**	**20**	**M**	**1092**	**Y**	**Y**	**Y**	**Y**	**Y**	**Y**	**Y**	**Y**	**GDs**	**-**	**CGs**
123	**Dhanalakshmi**	**39**	**F**	**1312**	**Y**	**N**	**Y**	**N**	**N**	**N**	**N**	**N**	**HH**	**-**	**NSP**
124	**Esther**	**40**	**F**	**1674**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
125	**Velaiyudam**	**57**	**M**	**1442**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Y**	**CaS**	**+**	**Ade c**
126	**Veerapan**	**30**	**M**	**1654**	**Y**	**N**	**Y**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**CGs**
127	**Narayanan**	**54**	**M**	**2245**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**DU**	**+**	**CPDs**
128	**Chandra**	**38**	**F**	**2432**	**Y**	**N**	**N**	**N**	**Y**	**N**	**N**	**N**	**HH**	**-**	**CGs**
129	**Sounder**	**40**	**M**	**3215**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
130	**Rajendran**	**57**	**M**	**2425**	**Y**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DU**	**-**	**CPDs**
131	**Murugan**	**73**	**M**	**3786**	**Y**	**N**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**CaS**	**-**	**Ade c**
132	**Alexander**	**67**	**M**	**3190**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
133	**Aravindan**	**47**	**M**	**1365**	**Y**	**N**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
134	**Senduran**	**31**	**M**	**2674**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
135	**Jaikumari**	**27**	**F**	**1321**	**Y**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**EGs**	**-**	**CGs**
136	**Preethi**	**19**	**F**	**1644**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**GERD**	**+**	**RE**
137	**Balagopal**	**65**	**M**	**1899**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**Gs**	**-**	**CGs**
138	**Shiva**	**35**	**M**	**3423**	**Y**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**N**	**Gs**	**-**	**ACG**
139	**Madhan**	**37**	**M**	**2323**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**N**	**Y**	**GERD**	**+**	**RE**

140	**Vijayan**	**57**	**M**	**1563**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Gs**	**+**	**HPG**
141	**Gowthaman**	**39**	**M**	**1765**	**Y**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**
142	**Sundari**	**48**	**F**	**5645**	**N**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Ds**	**+**	**CPDs**
143	**Malliga**	**24**	**F**	**2432**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**NS**	**-**	**NSP**
144	**Seetharaman**	**38**	**M**	**3864**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**HH**	**-**	**NSP**
145	**Renuga**	**35**	**F**	**2342**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**N**	**GERD**	**-**	**RE**
146	**David**	**38**	**M**	**2654**	**N**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**GERD**	**+**	**RE**
147	**Hemalatha**	**26**	**F**	**1177**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**N**	**N**	**NS**	**-**	**CGs**
148	**Savithri**	**66**	**F**	**3433**	**N**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**	**GU**	**+**	**ACG**
149	**Latha**	**32**	**F**	**1024**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**N**	**GERD**	**-**	**NSP**
150	**kalaiyarasan**	**38**	**M**	**1207**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**N**	**N**	**GERD**	**-**	**RE**

KEY TO MASTER CHART

S NO - Serial no

NSP – No specific pathology

OP NO - Out patient no

EE – Esosinophilic esophagitis

M – Male

RE – Reflux esophagitis

F – Female

ACG – Active Chronic Gastritis

Y – Yes

CGs – Chronic Gastritis

N – No

HPG – H.Pylori Gastritis

EP/D – Epigastric pain/discomfort

CPDs – Chronic Peptic Duodenitis

N/V – Nausea/Vomiting

Hyp P – Hyperplastic polyp

Es – Esophagitis

Gs – Gastritis

Ds – Duodenitis

EGs – Esophagogastritis

GDs – Gastroduodenitis

ESDs – Esophagogastroduodenitis

GU – Gastric ulcer

DU – Duodenal ulcer

HB – Heart burn **NS – Normal study**

Sq ca – Squamous cell carcinoma

FI – Food intolerance **HH – Hiatus Hernia**

Ade c - Adenocarcinoma

IDG – Indigestion **BE – Barett's esophagus**

Sig c c – Signet cell carcinoma

LW/A – Loss of weight/Appetite **EP – Esophageal polyp**

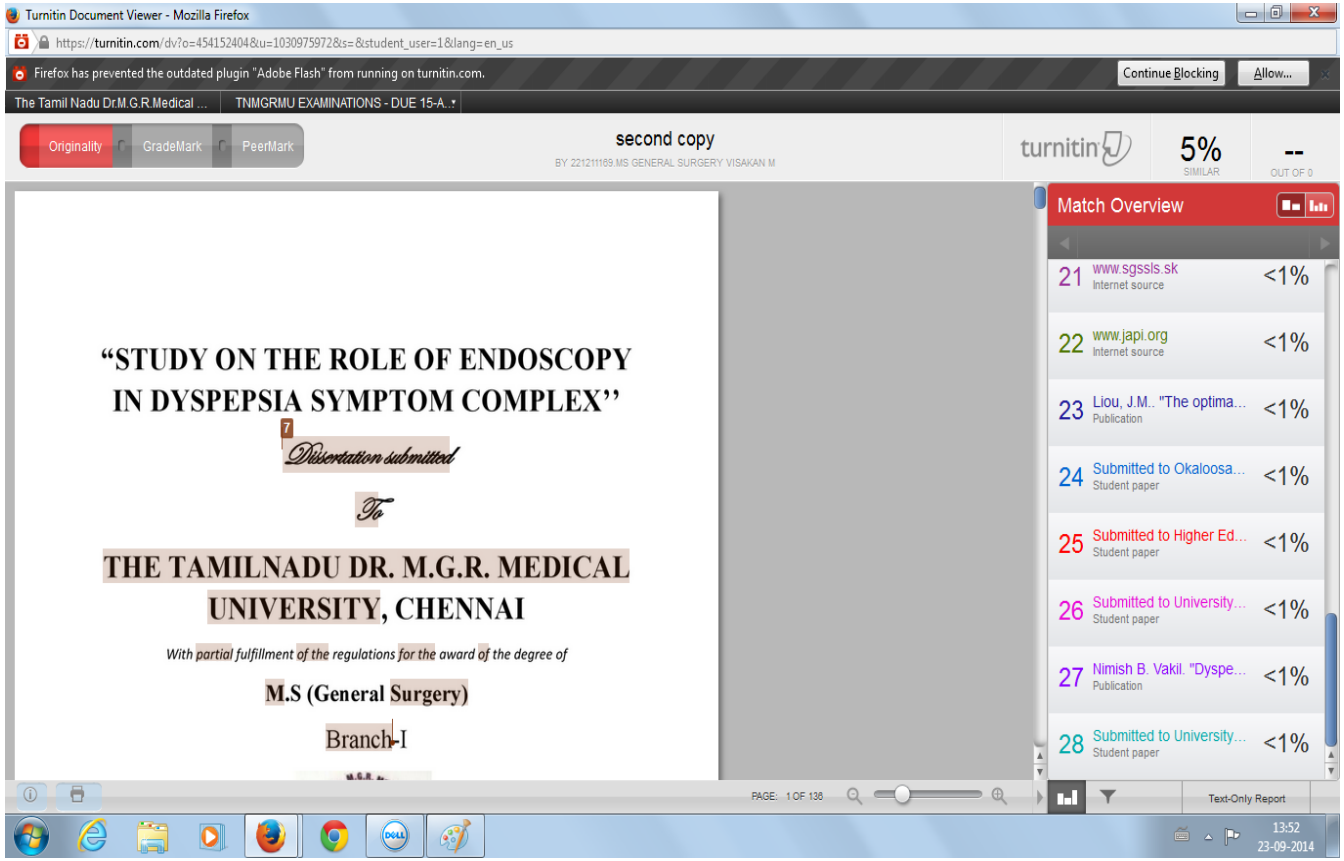
+ Positive **- Negative**

P – Pallor **CaE – Carcinoma**

ET – Epigastric tenderness **CaS – Carcinoma of stomach**

HPE – Histopathological examination

GERD- Gastro oesophageal reflux disease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INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10

Ref.No. 18520/ME-1/Ethics/2013 Dt:05.12.2013

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on Role of endoscopy in dyspeptic symptom complex - For Project work Submitted by Dr.M.Visakan, MS (GS), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information / informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
19 FEB 2014
Kilpauk, Chennai-10.

19/2/14.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,
Chennai